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EDITORIAL

CONSULTATIONS

THE general dentist, as well as the specialist, should make use of consultations more often, and consultants should cooperate more effectively with confreres who request consultations.

In the various departments of the Armed Forces official consultants are available, and their services are used whenever a patient presents problems of an unusual nature or when general medical conditions complicate the oral problems.

In private practice, consultations should be suggested to the patient before he loses confidence in the care he is receiving. Patients often feel embarrassed in requesting another professional opinion, and confidence is strengthened when consultations are suggested by the doctor.

Consultations are indicated if radical surgical procedures are contemplated, especially if the surgery will interfere with the patient's occupation or will change his appearance or functional ability. In such cases the consultant should, first of all, confirm the diagnosis, then give his opinion regarding prognosis, and pass judgment on the required treatment.

Consultations also may be advisable if the suggested procedures involve great financial sacrifice or if the result is a compromise between what the patient expects and what can be offered by the available therapeutic and restorative measures. By such consultations, the patient is given assurance that he is receiving the most efficient and most up-to-date treatment, and he has a chance to consider a compromise that will give adequate results.

Consultations also serve as a legal protection. They are an indication that the patient was well cared for. Call a consultant if you are doubtful about a diagnosis or if any aspect of the case is beyond your knowledge and experience. Call a consultant if the treatment that you have instituted does not give the expected result, if complications set in, or if during the course of the treatment you realize that the situation is more serious than it seemed at first. It has been pointed out by legal experts that few malpractice actions are lost on trial when an independent consultant testifies that he has been asked to examine the patient and to give advice.

Consultations are also advisable if the patient feels that he is not doing as well as expected, even though you are certain that your treatment is correct. It gives the patient confidence in your ability and assurance that his welfare is uppermost.

Medical consultations are particularly important. If it is suspected that some systemic factor is an underlying cause, or if a stomatologic condition causes complications in other systems of the body, a medical consultant should be recommended to confirm the suspected complications as well as to institute general therapeutic measures. One should always keep in mind that systemic conditions, when not recognized and controlled, will frequently prolong local therapy or invite therapeutic failure and recurrence of the disease.

In some cases one should insist on a consultation and, if the patient refuses to consent, a letter should be sent summing up your recommendation and his refusal. A carbon copy of such a letter or an entry on the hospital chart, properly witnessed, may become an important document in any litigation.

Consultants should always make written reports—on a consultation sheet if the patient is hospitalized or by letter if the patient comes to the office—so that the consultation can be added to the record. In order to get the best results, one should make available to the consultant the findings of special tests, the information obtained from x-ray studies and generally the x-rays themselves, and pathology reports if a biopsy has been taken (and often the microscopic slide should be furnished). The consultant should be at liberty to have additional x-rays made if he deems it necessary or to take a second biopsy if he feels that the original one is not satisfactory. As a rule, he should see the patient personally rather than examine only his x-rays, for physical examination and evaluation of the patient's attitude regarding both his disease and his doctor are important. The consultant should give an unbiased and impartial opinion.

If the patient is referred for diagnosis only, the afore-mentioned report ends the doctor's responsibility. If he is referred for medication or surgery, it should be so stated and the patient also should be so informed. It is not a sign of incompetence to refer a patient to a specialist for treatment. Everyone today realizes that there are special fields in which those who have had graduate education and extensive experience excel and therefore can render better service than that obtainable from those who limit themselves to the treatment of the much more common general and uncomplicated conditions.

While the report of the findings of the consultant should be made to the dentist who refers the patient, it is often advisable for the consultant to give the patient, with the dentist's consent, a brief statement regarding his condition, the reason for his recommendations, and the benefit that may be derived therefrom.

It is the patient who benefits most from a consultation, but consultations in addition serve to cement good relationships between the general practitioner and the specialist or between the specialist and his confrere who may have unusual knowledge in one area of his field or greater clinical experience. Discussions with consultants bring out valuable information regarding the advances which are constantly made in the diagnosis and treatment of disease. Consultations thus may be looked upon as an educational procedure—a refresher course in a special field.

K. H. T.

OPERATIVE ORAL SURGERY

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MYXOMATOUS TUMORS OF THE JAWS

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BECAUSE myxomas and fibromyxomas of the jaw are rare, descriptions of these tumors in the dental literaure usually have been in the form of individual case reports. The purpose of this article is to report on twenty-six lesions of this type seen at the Mayo Clinic and to give special attention to the correlation of histologic and clinical characteristics.

REVIEW OF LITERATURE

Differentiation From Other Types.—Some authors[4, 5, 8, 13] have pointed out that myxomatous areas exist within many benign and malignant tumors of mesenchymal origin. They said that these tumors must be classed with the group which their primary element most closely resembles histologically.

Willis[17] believed that myxomas and myxosarcomas are simply modified forms of fibroma and fibrosarcoma with mucin in the intercellular matrix.

Stout[13] thought that certain myxomas were true neoplasms. Ten of the forty-nine he reported were bony lesions, five of them being in the mandible, three in the maxilla, one in the clavicle, and one in a metatarsal bone. He stated that a majority of the bony myxomas are found in the jaws.

Some authors[5, 8] have disregarded myxomatous tumors of bone in their classification. Lichtenstein[8] declared: "The designation of 'myxoma' has been applied to certain tumorlike, myxoid connective tissue lesions in the jawbones particularly, although some of these lesions at least may be plausibly regarded as peculiar modified foci of fibrous dysplasia. Altogether, I have never encountered a skeletal neoplasm which warranted the designation of pure myxoma or myxosarcoma of bone."

One of us (D. C. D.), on consideration of 2,276 primary tumors of bone at the Mayo Clinic, found no myxomas in bones outside the facial skeleton.

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Origin.—Thoma,[14] discussing etiology and classification of myxomatous lesions of the jaw, grouped these tumors into osteogenic and odontogenic types. The more common odontogenic tumors, he said, were almost always benign, and the osteogenic tumors were most often malignant, like myxomatous lesions in other bones.

Thoma and Goldman[15] concluded from their study of eleven cases that the tumor was benign and of odontogenic origin. They suggested that an odontogenic fibroma developing from the mesenchymal portion of the tooth germ (dentine papilla, dental follicle, or periodontal membrane) could undergo differentiation or mucoid degeneration of the fibroma to form a myxoma. Ten of their eleven cases were found in the toothbearing part of the jaw and were associated with dental abnormalities. Bernier[1] was in essential agreement and discussed odontogenic myxoma and fibroma together. Harbert and associates[6] said that myxomas developed from embryonal rests. Bruce and Royer[2] doubted that myxomatous tumors represented a degenerative process in a previously existing fibroma. They thought that, at least hypothetically, the tumor could be accounted for by the proliferation of mesenchymal rests within the alveolar bone.

Age and Sex.—Among reported cases, males and females were affected with almost equal frequency. Seldom was the patient younger than 10 years or older than 50 years; almost 67 per cent of the patients were between 10 and 29 years of age.

Location.—Almost two-thirds of the lesions described in the literature were in the mandible. Within the mandible the angle of the jaw, the ramus, and the adjacent molar region were most commonly affected, but some tumors extended anteriorly. A few affected primarily the anterior portion, but bilateral tumors were rare. Within the maxilla, the zygomatic process and alveolar bone bearing the molar and premolar teeth were affected most often. Frequently the sinus was involved, especially by larger tumors. Tumors of the anterior maxilla were less common.

Signs and Symptoms.—The sign most frequently reported was a slowly progressive swelling and facial deformity, usually painless. Thoma and Goldman[15] mentioned pain as a chief complaint, while Bernier[1] stated that absence of pain was noteworthy. The teeth in the affected region often were malposed, but more frequently they had been extracted in an attempt at treatment. Some teeth had been loosened because of destruction of the supporting bone. The mucosa was normal except for occasional ulceration secondary to biopsy or dental extraction. These signs and symptoms had been noted for periods varying from one week to eight years, usually from one to three years, before treatment was begun.

Roentgenologic Diagnosis.—The typical roentgenologic appearance of the tumor was described as a multilocular, radiolucent area with well-defined, sclerotic margins. A few lesions were unilocular. On extension of the lesion into the alveolar bone, the margin sometimes became scalloped between the

roots of the teeth.[14] Although some authors stated that the tumor showed no trabeculation, Sonesson[11] used the presence of gracile, straight trabeculae to help distinguish this tumor from cysts and ameloblastomas as well as from fibromas and even fibrous dysplasia, which are extremely difficult to differentiate by roentgenologic means.

The mandible bearing a tumor often was expanded; the cortex was sclerotic and usually intact. In the maxilla the tumor invaded not only the bone but the antrum as well, appearing as a large, rounded mass of soft tissue on the anteroposterior or Waters' views. Occasionally the antral walls were destroyed by the tumor.

Invasion of soft tissue is more characteristic of ameloblastomas than of myxomas. The tumors were reported to vary from 1 to 10 cm. in diameter. Most authors[7, 11, 16] agreed that cyst, fibroma, ameloblastoma, giant-cell reparative granuloma, and fibrous dysplasia must be included in the differential diagnosis because of their similar roentgenologic appearance. Hovnanian[7] thought that the diagnosis of sarcoma must be entertained, even though the lesion appears roentgenologically benign.

Pathologic Diagnosis.—The contour of the cortical bone was reported to be distorted because of the expansion of the tumor beneath it. The bone usually was intact,[11] giving a firm, even solid, feel to the lesion. The overlying mucosa was intact and normal in color, only occasionally slightly hyperemic.

On exposure, the myxoma itself presented a bosselated surface and was usually soft. Occasionally the surface was covered by glistening mucus. Fibromyxomatous tumors usually were firmer in consistency. The color was described as grayish white, milk white, yellowish, or even amber. Some authors[14] have stated that the myxoma was encapsulated; others[4] stated specifically that encapsulation of these tumors was rare.

The cells usually were stellate, but occasionally they were triangular or cuneiform. Their cytoplasmic processes were long and anastomosed with one another throughout the mass. Variable numbers of normal-appearing fibroblasts were found. Willis[17] and others[14] believed that the myxomatous cells were derived from the fibroblasts by degeneration. The cytoplasm of the stellate cells stained poorly but was usually basophilic and finely granular. The nuclei of the myxomatous cells were ovoid and hyperchromatic; few mitotic figures were seen. The intercellular substance was mucus, but occasionally areas of collagen appeared. Cystic areas and areas of hemorrhage were found sometimes. In addition, scattered lymphocytes, plasma cells, and large mononuclear cells were seen. Some authors[4, 9, 12] reported strands of inactive-appearing epithelial cells, probably of odontogenic origin, within the tumor.

Treatment.—A combination of simple enucleation and curettage has been credited with therapeutic success, but most authors cautioned that this must be done thoroughly if recurrence is to be avoided. Other workers removed the major portion of the tumor with a curette and followed this with chemical or electrical cautery. Excision en bloc, retaining the continuity of the bone, was suggested by many as the best method of treatment. Resection of the tumor

and involved bone has been reserved by most surgeons[1, 15] for extensive lesions or tumors which have recurred after more conservative treatment. Radiation, according to most authors,[1, 7] has no place in the treatment of these tumors, since they are not radiosensitive.

Prognosis.—Myxomas and fibromyxomas of the jaw were found to be persistent lesions and most difficult to eradicate; however, like the ameloblastomas, they were benign. Stout[13] declared that metastasis of myxomas never has been reported except in tumors of the heart, and other authors[1, 6, 7] likewise said that metastasis does not occur. Most authors agreed that sarcomatous change eventuates rarely, if ever.

PRESENT STUDY

Materials and Methods.—The registration numbers of all patients with a diagnosis of myxoma (or any tumor in which the descriptive term *myxo-* was used) of the jaw were taken from the pathologic registry at the Mayo Clinic for the years 1907 through 1956. Only specific designation of the lesions as myxomatous polyps caused rejection at this stage. The search yielded forty-five cases for consideration.

The gross specimens were studied, and all available microscopic slides were reviewed. When it was thought necessary, additional slides were prepared from the preserved gross specimens. By this means twelve malignant tumors were eliminated from the group, with the diagnosis of fibrosarcoma in five instances and osteogenic sarcoma in seven. Also, seven benign lesions (two nasopharyngeal fibromas, two inflammatory lesions, one cyst, one fibroma, and one lymphangioma) were eliminated. There remained a group of twenty-six tumors which were primarily myxomatous or fibromyxomatous.

An attempt was made in this early phase of the study to divide myxomatous tumors on a histologic basis alone into benign and malignant forms. The criteria used were (1) the number of binucleate cells, (2) the number of mitotic figures, (3) the degree of cellularity, (4) anisocytosis, and (5) the hyperchromatic appearance of the nuclei. The first three criteria were of little value. Binucleate cells were common enough in all tumors to eliminate this feature as a criterion for malignancy; mitosis rarely was found, even in the most active tumor; and the number of cells was likewise of little value because of the variation from very cellular to acellular areas, even within the same lesion. Furthermore, only a vague impression of activity was gained by considering these three characteristics together. The criterion which seemed most valuable to us was anisocytosis. This condition, together with the hyperchromatic appearance of the nuclei, formed the only basis on which we could separate the histologically benign and malignant forms. Twenty of the lesions were classed as benign on the basis of their cellular characteristics alone (Fig. 1, *a*), and six appeared initially to be malignant. All of these six supposedly sarcomatous forms were judged to be of a low grade of malignancy (Fig. 1, *b* and *c*), and some of the other tumors were called benign only after considerable debate because of their similarity to the malignant forms.

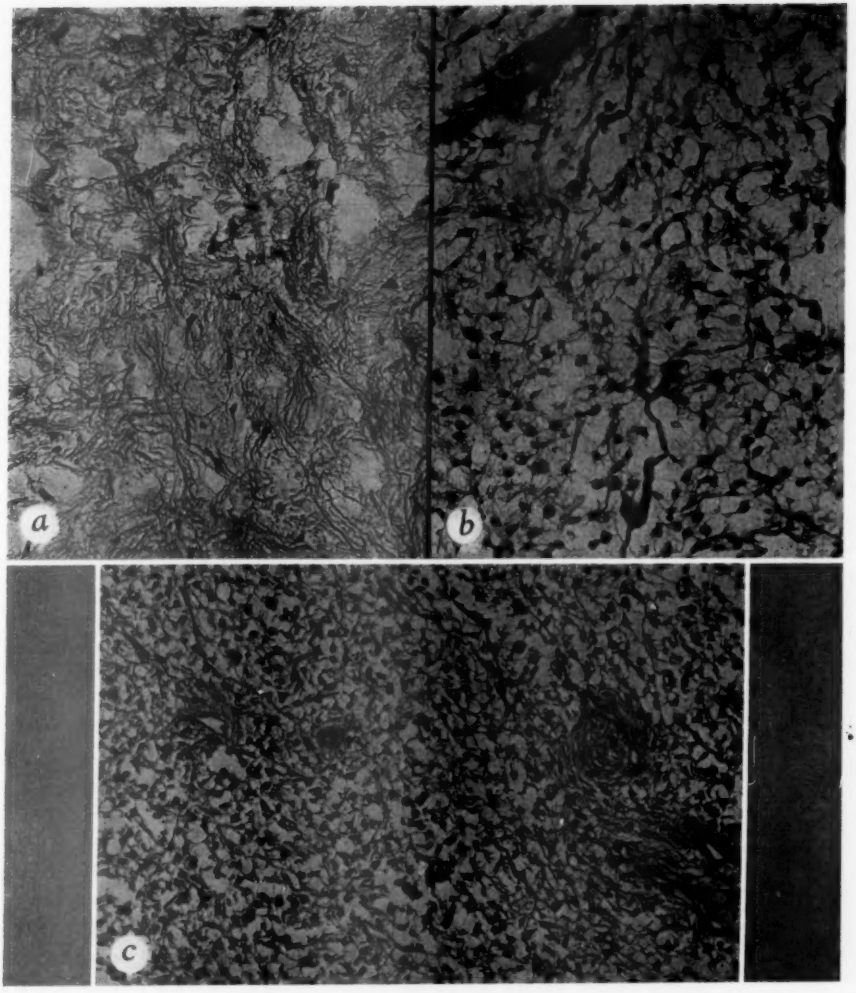

Fig. 1.—Microscopic sections. *a*, This inactive-appearing lesion of the maxilla was 2.5 cm. in diameter when initially curetted. It recurred three years later and required more extensive therapy. *b*, This more active-appearing tumor was recurrent when seen at the clinic and was considered malignant. The treatment included excision, electrocoagulation, and roentgen therapy. It had not recurred in eleven years. *c*, This active-appearing tumor, considered malignant, was one of several small yellowish masses of softer consistency within a tumor of the maxilla which otherwise appeared histologically benign. It had been curetted two years before the patient was seen at the clinic, and after a second curettage there it recurred again in six months. The patient is alive and well with no recurrence five and one-half years after the third curettage. (Hematoxylin and eosin stain. Magnification, ×175.)

These categories will be used in the present report, although they are not supported by some other observations made.

After this histologic selection of the group, the histories, follow-up information, and all available roentgenograms were reviewed.

Age and Duration.—Of the twenty patients whose lesions appeared histologically benign, the average age when they were first treated at the clinic was 30 years and the median age was 29 years (Table I). The range of their ages was 13 to 64 years. The ages when these patients first noted the tumor averaged 26.5 years and ranged from 6 to 64 years. Only one patient noted the lesion before he was 10 years old. The average duration from onset to the first treatment at the clinic was 4.7 years but, because of previous treatment elsewhere in some cases and especially long duration in a few, the median of one year probably better indicates the duration of symptoms. The range of duration was from a maximum of twelve years for one patient to discovery while another was undergoing examination at the Mayo Clinic.

TABLE I. AGES OF TWENTY PATIENTS AT ONSET OF SYMPTOMS AND AT TIME OF FIRST TREATMENT AT CLINIC

AGES (YEARS)	AT ONSET OF SYMPTOMS (NO. OF PATIENTS)	AT TIME OF TREATMENT (NO. OF PATIENTS)
0- 9	1	0
10-19	5	4
20-29	6	6
30-39	7	5
40-49	0	4
50-59	0	0
60-69	1	1

The six malignant-appearing lesions were present in patients aged 22 months and 15, 16, 30, 47, and 61 years, and they had been noted for periods of one week, two years, seven years, three years, four months, and one year, respectively.

Signs and Symptoms.—The tumors caused slowly progressive swelling of the affected jaw and, in some cases, severe facial deformity. Maxillary tumors often filled the antrum and caused exophthalmos. Frequently teeth were displaced or tipped, but in many cases they had been removed previously. The cortical bone was expanded and thinned. The mucosa was normal or only slightly hyperemic except in one case in which the patient had ulceration with secondary infection following biopsy and electrocoagulation elsewhere. One patient, who had undergone extensive treatment in the past, had anesthesia of the affected side of the mandible. Slight pain was described in five cases. Trauma was associated in only two cases.

Two of the six lesions classed histologically as malignant had grown rapidly. In general, however, no clinical distinction could be made between the so-called malignant and benign tumors.

Sex.—Ten of the benign lesions were in female patients and ten were in male patients. Of the six malignant lesions, four were in males and two were in females.

Location and Size.—Ten of the benign tumors were found in the maxilla and ten were found in the mandible. Most of the benign lesions of the maxilla had expanded beyond this thin bone. Four of the ten were found at operation to fill the sinus and distend its walls; and four were found primarily in the premolar and molar region of the maxilla, having invaded the sinus to various degrees. Two lesions involved only the anterior region of the maxilla, and both of these extended across the midline. Expansion of the alveolar bone often caused malposition of teeth, but usually the greatest deformity was in the canine fossa or zygomatic area. The smallest maxillary tumor measured 2 by 2 by 3 cm., and the largest measured 8 by 9 by 6 cm.

The mandibular tumors involved the ramus and body of the bone with equal frequency. Two tumors involved the entire mandible from midline to condyle. One extended from the premolar region on one side to the premolar region on the opposite side. The anterior part of the mandible was less frequently involved than the posterior part of the body and the ramus. The angle of the jaw was affected by six of the lesions. The smallest mandibular lesion was 2 by 2 by 1 cm. and the largest was 6 by 5 by 4 cm.

The six so-called malignant lesions were somewhat larger than the benign ones. Five were found in the maxilla and one was found in the mandible.

Roentgenologic Diagnosis.—All available roentgenograms were reviewed, and our observations were in general agreement with those published by others. No roentgenologic distinction was found between the histologically benign and malignant examples. The tumors of the mandible most commonly were multilocular and cystic. Two tumors appeared to be unilocular but were cystic. Three tumors were characterized as "mottled." (That is, in addition to the fine trabeculae within the tumor, small calcified masses, either round or oblong, were seen.) These lesions easily could be confused with fibrous dysplasia. All of the tumors were well circumscribed and appeared to be slowly growing, expansile lesions. Only if the tumor had reached great proportions was the cortex of the bone perforated.

Some roentgenograms of the maxillary tumors showed only clouding of the antrum; others showed a mass of soft tissue invading, sometimes filling, the sinus. The walls of the antrum were expanded but seldom destroyed. The dental films clearly showed alveolar involvement (Figs. 2 and 3).

Pathologic Findings.—Our histologic findings agreed in essence with those of other authors. We believed that the basically myxomatous tumor could be differentiated from fibroma and fibrous dysplasia by the paucity of collagen within its central portion. Several of the tumors exhibited epithelial strands, similar histologically to dental epithelium.

Several of these myxomatous tumors contained slightly to prominently fibrous zones, suggesting the possibility of a relationship to fibroma. The most arresting feature of all of them, however, was the component resembling exactly that of myxomas of the somatic soft tissues. Whether these predominantly myxoid tumors are actually related to fibromas or other lesions is of academic importance only.

Treatment.—The treatment of the twenty lesions designated benign was usually curettage. The mass was enucleated if possible, and the bony cavity was carefully explored for residual tumor. In eight of the cases electrocautery

Fig. 2.—Unusual roentgenographic appearance. *a*, Myxomatous lesion, 8 by 6 by 4 cm., of the left maxilla, which appears partially calcified. It had been treated unsuccessfully with radiation for six years before the patient was first seen at the clinic. Here it appears more like fibrous dysplasia than a myxomatous tumor. *b*, This mandibular tumor was histologically a pure myxoma.

also was applied to the base of the tumor or within the bony cavity if it was difficult to determine whether all the tumor had been removed. For three of the tumors, external irradiation and radium therapy were added to the treatment described. Only one patient was treated by excising one side of the

mandible. The tumor in this instance was considered malignant at the time of operation, for it was a recurrent lesion when the patient was first seen at the clinic.

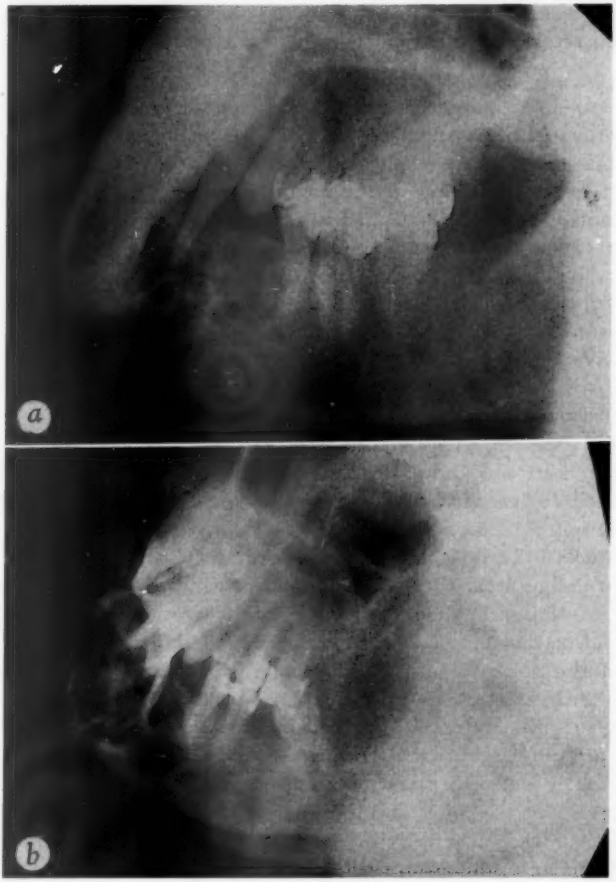

Fig. 3.—*a*, This recurrent mandibular lesion, which had been curetted ten years before it was first seen at the clinic, was treated by dental extraction, curettage, and marsupialization. Four years postoperatively there was no recurrence. *b*, A multilocular cystic appearance is typical of myxomatous tumors, but bilateral involvement in the anterior portion of the mandible is uncommon. Usually this type of lesion is found at the angle of the jaw.

Three of the patients underwent operations more than once at the clinic; one patient was treated four years after the initial operation and again two years later, and two others were operated on two and three years, respectively, after the original operation. All three originally had rather large maxillary lesions.

Of the six lesions considered histologically malignant, five were believed by the surgeon to be malignant at the time of treatment and were treated more

radically for this reason; but most of them were larger than the benign lesions and, although they were treated more extensively, they were thought too invasive to be removed completely. Consequently, these five were treated by curettage and cautery, followed by irradiation. The remaining tumor, an encapsulated mass, was enucleated, and the site was cauterized; the patient in this case had received irradiation therapy over a period of six years before operation.

Prognosis.—The prognosis for the benign lesions is excellent in the sense that the patient's life is not jeopardized and no malignant changes in bony lesions of this type have been demonstrated. However, even the benign lesions are difficult to eradicate and, unless completely removed, recur rapidly. None of our patients is known to have died as a result of the tumor.

In the twenty cases of tumor considered benign, five of the lesions were recurrent when first seen at the clinic. The previous treatment in these five cases was surgical and had been performed three months and one, three, four, and ten years previously. Of the twenty patients, we were able to trace eleven for five years or longer after treatment at the clinic. One of these had a recurrence treated elsewhere three years after operation here. Addition of the previously mentioned three patients operated on more than once at the clinic in less than five years makes a total of fourteen patients accounted for, among whom four are known to have had lesions recur after treatment here. The percentage of recurrence of these histologically benign tumors after treatment at the clinic thus was comparable to the percentage of lesions considered recurrent when first seen—that is, approximately 25 per cent. Tumors recurred in periods ranging from three months to ten years, usually two to three years.

Three of the six patients considered to have malignant lesions had had surgical treatment before being seen at the Mayo Clinic.

One of the patients with a lesion originally diagnosed as a fibromyxosarcoma died with extensive local recurrence two and one-half years after the first of six surgical and radiologic attempts at cure. The cause of death was said to be a "lung condition." Biopsy specimens of this tumor at the second, third and fourth surgical procedures appeared histologically like fibrosarcoma; however, review of the slides and the first surgical specimen removed forced the conclusion that when first seen the tumor was basically a myxosarcoma. This is the only tumor of the group which conceivably could be considered to have metastasized, and the evidence is only presumptive.

Of the five remaining patients whose tumors were thought malignant, one had a recurrent tumor removed six months after operation at the clinic but is alive and well without further recurrence six years after initial treatment. Two other patients are alive and well without recurrence five and eleven years after treatment at the clinic. Follow-up information was not available on the two remaining patients.

COMMENT

Since myxomas and myxofibromas occur almost exclusively in the tooth-bearing portions of the jaws, since the myxomatous tissue histologically

resembles the stellate reticulum found in developing teeth, and since the lesions behave clinically much like the ameloblastoma, it seems reasonable to assume with authors cited earlier that these tumors are of odontogenic origin.

We believe that true myxomas must be extremely rare in long bones, if they occur there at all, and that lesions in long bones reported as myxomas actually are fibroma, fibrous dysplasia, fibrosarcoma, chondrosarcoma, osteogenic sarcoma, or other tumors of mesenchymal origin containing myxomatous elements, all of which were excluded from our study. Basically myxomatous tumors do occur in the jawbones, and almost always we found them clinically benign although recurrence is not uncommon, especially if removal of the lesion is not complete.

The tumors considered malignant usually were too large to be extirpated as thoroughly as those considered benign but, even so, recurrence was not invariably observed. These two facts suggest that the tumors actually were more benign clinically than was suggested by their histologic characteristics.

We think that the benign and malignant tumors (if malignant forms of myxoma exist in bone) cannot be differentiated with certainty on a histologic basis. Willis said: "The attempt to distinguish benign myxomas and myxosarcomas is even more futile than with their collagenous counterparts." In our own study, when roentgenologic, historical, surgical, and follow-up data were considered, the original division into benign and malignant tumors was found to be of doubtful significance. Unfortunately, follow-up information was not present in enough of the histologically malignant cases to validate this conclusion.

Most authors agree with our observation that the tumor is aggressive and difficult to eradicate, but that it is benign and does not metastasize.[6, 7] Certainly this is corroborated by the fact that only one of our cases presented the slightest question of metastasis.

Because of the extensive local involvement and the tendency to recur, the tumor must be vigorously treated. One surgeon expressed the opinion that block excision within the continuity of the bone, when feasible, was probably the best form of treatment for these tumors, and both he and the roentgenologist who worked with him decided that radiation was of no value in treating myxomas and fibromyxomas.

Our judgment, however, is that resection is indicated as a first measure only in cases of massive lesion.[1] For the recurrent tumors, more radical treatment may be necessary. We agree that irradiation is probably of no value in the treatment of these tumors,[1, 7] and we now know that irradiation of benign tumors of bone is potentially hazardous.[10]

SUMMARY

This study of twenty-six myxomas of the jawbones emphasizes that such tumors may be locally invasive and may recur after conservative management, but that they rarely, if ever, metastasize.

Since myxomas are practically unknown in bones outside the jaw, and since they resemble portions of the dental follicle, they are likely of odontogenic origin.

Their histologic appearance correlates poorly with the capacity of myxomatous tumors of the jaws to recur.

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MALAR FRACTURES

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IT IS very easy to overlook malar and zygomatic arch fractures in the absence of a routine procedure for the examination of facial injuries. We all agree that the initial examination made on any injured patient should be a general evaluation of his immediate condition. This is of vital importance in the handling of head trauma, for skull fracture (Fig. 1) or intracranial bleeding may place the patient's life in jeopardy.

NEUROLOGICAL SIGNS OF CRANIAL INJURY

It is not necessary that the oral surgeon be able to make a complete neurological survey of patients with facial fractures. He must, however, have sufficient knowledge to recognize the need for a neurological consultation. Some of the signs and symptoms of neurological injury presented below may be of help in evaluating the injured patient.

The most important sign, and the one which should be elicited first, is whether the patient experienced a loss of consciousness at any time after the accident. If the patient was unconscious and then regained consciousness, only to become stuporous again, intracranial bleeding is to be suspected. If the intracranial pressure rises, coma results. As the pressure is reduced, the patient becomes more rational. Therefore, the best index of the patient's condition is the degree of orientation and rationality.

Headache, dizziness, nausea, and vomiting are not to be taken lightly and may be indicative of serious brain injury. Diplopia following a fracture in the walls of the orbit may also be due to cranial injury of a more serious nature. A comparison of the size of the pupils will elicit useful information. If they are unequal in size, or if one pupil is ragged, this should be noted. Bleeding from the ear should not be investigated by disturbing the blood clot, since this may be an indication of a skull fracture in the middle cranial fossa. The nasal discharge of serous fluid tinged with blood or nasal bleeding may be due to fracture of the cribriform plate of the ethmoid.

Considerable force is necessary to fracture one of the facial bones. Contusion, laceration, or other injury to the cranial contents usually occurs on the

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side of the head opposite the initial impact. Trauma thus transmitted through the bones of the skull produces inflammation, laceration, or edema of the meninges or brain. The resultant increase of fluid within the bony walls of the skull raises the intracranial pressure.

Patients do not die from the presence of a skull fracture; rather death comes as a result of the increased intracranial pressure which depresses the medulla and thus directly affects the heart rate, respiration, and blood pressure. It is not necessarily true that brain damage is more extensive in cases of skull fracture. The reverse may well be true, for the soft tissue of the cranial contents is more susceptible to injury which drives the brain into the protuberances of the cranial floor.

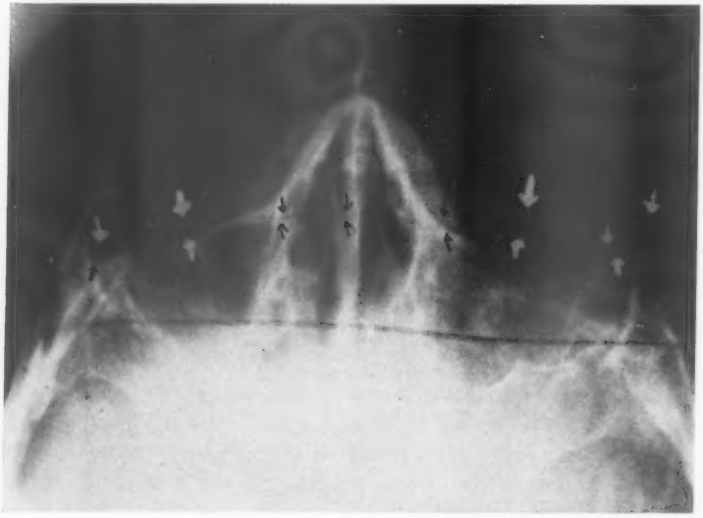

Fig. 1.—A case of severe cranial injury with associated skull fracture.

An elevation of the blood pressure with a pulse rate below 60 is significant. A sudden increase in pulse rate with a fall in blood pressure is a very grave sign. These symptoms are the result of stimulation of the cardio-inhibitory and vasomotor centers in the first case and the exhaustion of these centers in the second case. Daily fluctuation of temperature is common in brain injuries, but irregular respiration is a very grave sign.

It is thought that the peak of reaction following brain injury occurs within the first two hours. It would be best not to treat any patient with a head injury within the first twenty-four-hour period. Certainly, one should wait an even longer period before manipulating any facial fracture which may have proximation to a skull fracture. There is rarely any emergency necessitating immediate manipulation of facial fractures; therefore, treatment can be put off for ten days to two weeks in the presence of cranial injury.

The x-ray diagnosis should be secondary to the clinical examination for the presence of increased intracranial pressure. Should these signs be present, then it is more important to direct treatment to the amelioration of this condition than to proceed with treatment of the facial injury. The manipulation required for an x-ray examination may increase the danger to the patient, therefore, this should not be undertaken without the neurologist's consent.

EXAMINATION

Facial trauma is not always limited to the simple fracture of one bone. Considerable force is required to fracture the malar bone. Therefore, the first examination should be directed toward the evaluation of the patient's

Fig. 2.—Case 1. Severe orbital edema following facial trauma.

general condition. In the presence of any positive signs of cranial injury, the procedure outlined previously should be followed. In the absence of any positive neurological finding, the procedure suggested below is followed.

History of facial trauma with orbital edema should make the examiner suspicious of a malar fracture (Figs. 2, 7, and 17). The clinical examination should include a manual palpation of the rims of both orbits. Any depression in the floor of the orbital rim or lateral wall is usually apparent. Most fractures of the orbit can be felt through the facial edema if displacement has occurred. This can be further confirmed by an intraoral exploration along the maxillary wall and under the zygoma. In severely comminuted fractures, severe pain will follow the exploration and sharp bony edges may be felt in the fractured maxillary wall.

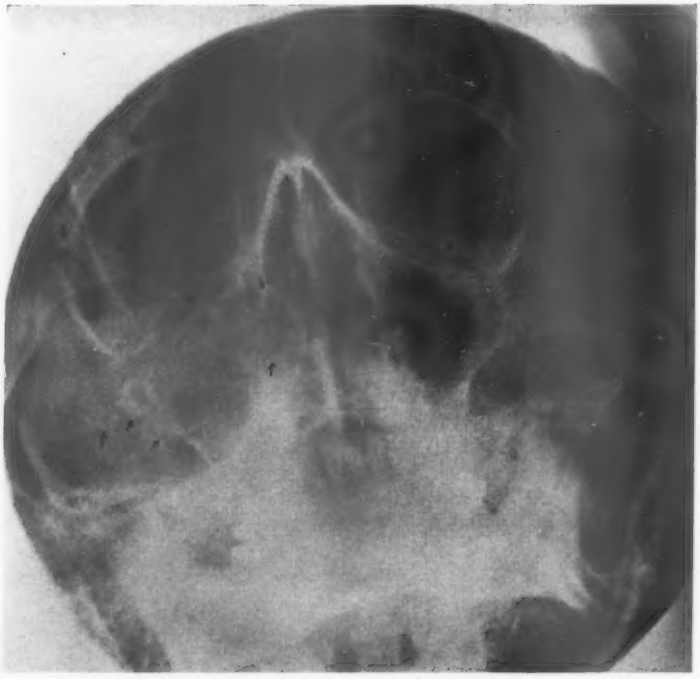

Fig. 3.—Case 1. Waters' position film showing fracture of the malar bone indicated by arrows.

The examination should be carried one step further to rule out or confirm the presence of maxillary fracture (Fig. 18). The maxilla may be fractured through the palate and have an additional fracture horizontally through one or both sides of the lateral wall. If this should prove the case, then the operative procedure to reduce the maxilla takes precedence over the reduction of the malar fracture. If these additional injuries can be treated with rubber-band traction, the rubber bands should be placed as early as possible. In some of these cases the maxilla may be impacted upward and backward, and

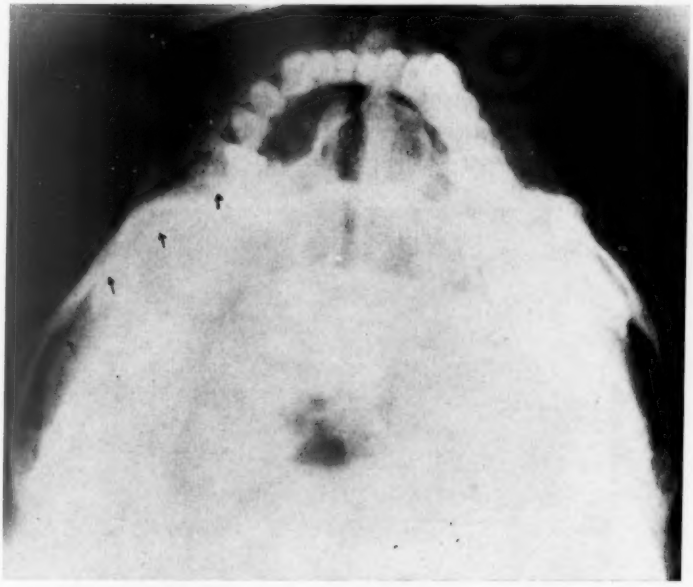

Fig. 4.—Case 1. Submental vertex view showing fracture of malar bone as indicated by arrows.

Fig. 5. Fig. 6.

Fig. 5.—Case 1. Drawing of the submental vertex view showing position of the fragments.

Fig. 6.—Case 1. Elevation of the malar bone through simple intraoral incision beneath the arch.

therefore downward, as well as forward, traction is required before fixation. If the maxillary fracture is pyramidal into the base of the orbits (Fig. 18), even more care must be exercised in the reduction because of the possibility of a fracture of the cribriform plate. In any case, it is beyond the scope of this article to discuss the treatment of these fractures, other than to point out the necessity for recognition of these additional possibilities (Cases 2 and 3).

Fig. 7.—Case 2. Severely lacerated face with orbital involvement.

We should suspect the type of fracture discussed above if there is movement of the entire maxilla or parts of the maxilla. On the other hand, conjunctival hemorrhage with infraorbital edema and paresthesia along the distribution of the nerve are almost always indicative of a fracture of the malar bone. It is not possible for these signs to exist without some displacement in the other areas of attachment of this bone.

If all signs of fracture are negative in the maxilla, it is still possible that there has been an injury to the zygomatic arch itself. There are instances in which depression may be palpated but in which there is no hindering of

mandibular movement. It is also obvious that the coronoid process of the mandible will be blocked by an arch which is depressed sufficiently to prevent its movement forward.

These are the clinical symptoms and the process of examination for diagnosis of fractures in the malar bone area.

Fig. 8.—Case 2. Submental vertex view of fractured malar bone and arch.

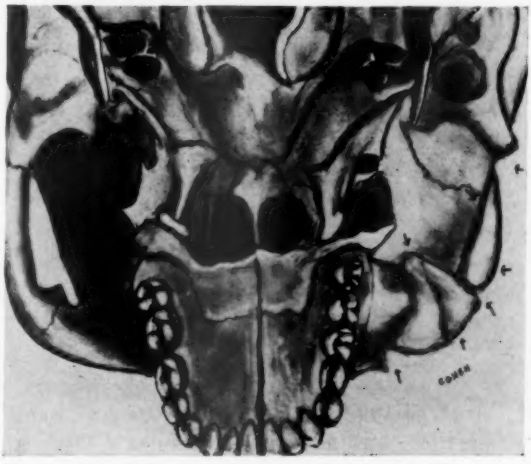

Fig. 9.—Case 2. Drawing of submental vertex view to show fracture site.

X-RAY EXAMINATION

Once the x-ray examination is permitted, it should be all-inclusive. The examination should be directed toward finding any facial fracture which may be present, as well as the malar fracture.

The following roentgenograms are requested: (1) Waters' position film (Figs. 3, 10, and 18), (2) right and left lateral jaw films, (3) posteroanterior view of the mandible, and (4) submental vertex film (Figs. 5, 8, and 19).

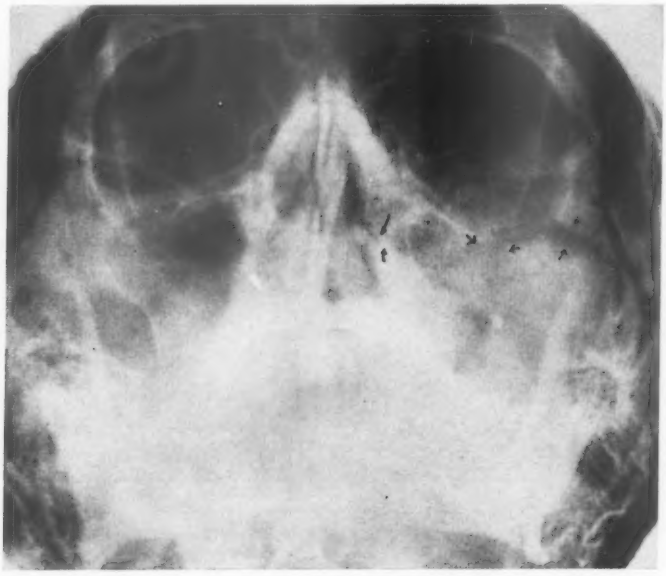

Fig. 10.—Case 2. Waters' position view showing severe maxillary and malar fracture.

If these initial films suggest the presence of other fractures, then additional x-ray pictures may be ordered at that time. It is a common problem in various small hospitals to experience some difficulty in obtaining these views. This is due to the relative infrequency with which personnel in these hospitals are called upon to make such an x-ray survey. Those who have been faced with this problem a number of times usually find it necessary to learn to use the medical unit and are able to help the technician obtain the films required.

CLASSIFICATION OF MALAR FRACTURES

This article will deal only with fractures of the zygomatic arch and the malar bone. It should be pointed out, however, that in many instances these will occur in conjunction with maxillary fractures. When this is the case, it is necessary to treat the maxillary fracture first and then turn to the treatment

of the zygomaticomalar fracture. For the purpose of discussion, once a diagnosis is established, the fracture may be one of the types listed below:

1. Simple fracture without displacement
2. Simple fracture with slight displacement but not sufficient to require surgery
3. Simple fracture of zygomatic arch of the depressed type; not associated with malar fracture

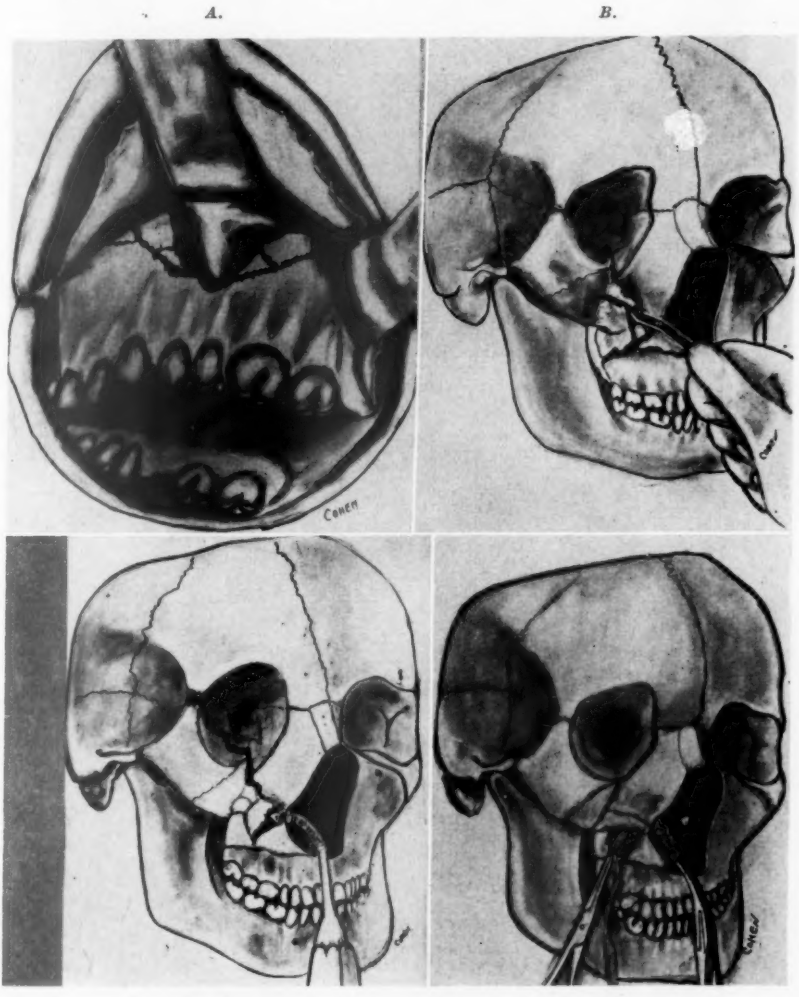

Fig. 11.—Case 2. *A*, Proper elevation of maxillary flap. *B*, Opening into the antrum to expose contents. *C*, Insertion of nasal rasp through nose into the antrum. *D*, Antrum packed with oiled tape.

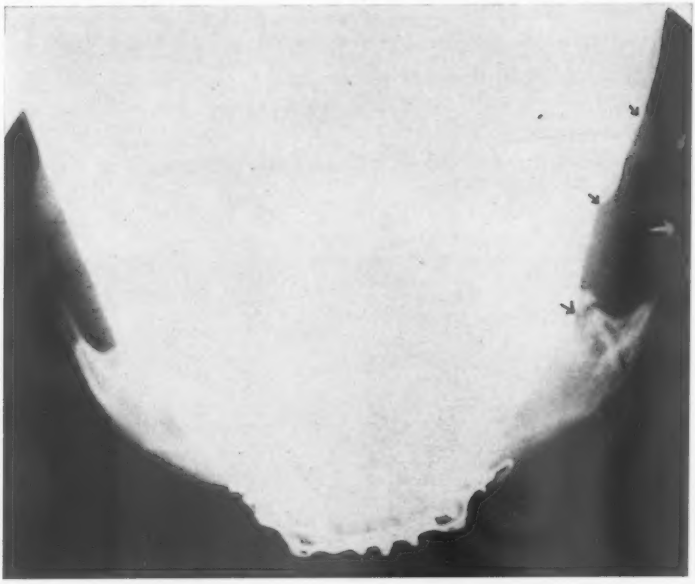

Fig. 12.—Case 2. Postoperative view of the reduction.

Fig. 13.—Case 2. Postoperative Waters' position.

4. Malar fracture with displacement
   (a) Upward
   (b) Downward
   (c) Medial
   (d) Posterior
   (e) Anterior
   (f) With zygomatic arch fracture

Fig. 14.—Case 2. Protective device placed after the reduction of the fracture.

Fig. 15.—Case 2. Intraoral cast buccal arcs ligated to reduce maxillary fracture prior to the reduction of the malar injury.

There are many possible combinations of the fractures just outlined. The criterion which determines the type of injury is related to the direction from which the trauma is delivered. For example, if the trauma is delivered from a lateral aspect, the fracture will be of the compressive type which drives the malar bone and the zygomatic arch medially into the region of the antrum. On the other hand, if the trauma travels posteriorly, it will fracture the malar bone upward and somewhat medially.

Fig. 16.—Case 2. Postoperative view of patient three months later.

ANATOMIC CONSIDERATIONS

To understand the injury that can result from this type of fracture, it is necessary to consider the contents of the infratemporal fossa. Any compressive injury which drives the malar bone into this area will, of necessity, involve some very important anatomic structures.

The masseter muscle has two points of origin on the zygomatic arch: (1) a deep origin along the entire length of the arch and (2) a superficial origin along the anterior two-thirds of the lower border. The coronoid process may be

blocked by compression of the arch and thus also prevent function. Compression of the lateral portion of the temporal muscle can cause limited movement of the pterygoids which are beneath it. Fracture in the arch will cause pain when the mandible functions, due to the inflammatory process in these muscles.

Fig. 17.—Case 3. Severely injured patient following automobile accident.

The infratemporal fossa contains the pterygoid muscles, the sphenomandibular ligament, the pterygoid venous plexus, the first and second parts of the maxillary artery, the mandibular nerve and its branches, the chorda tympani, the tendon of the temporalis, the coronoid process, and the masseter muscle. The foramen ovale and spinosa, which transmit the cranial nerves, also open into this fossa.

The pterygopalatine fossa and the pterygomaxillary fissure, with the various structures which pass through them, are also in danger of injury when trauma affects this area.

Neurological damage to the distribution of the infraorbital nerve is common because of fracture of the infraorbital canal associated with fracture of the malar bone. The nasal and labial paresthesia is rarely permanent, but on occasion the edema may extend into the facial canal and cause a temporary type

Fig. 18.

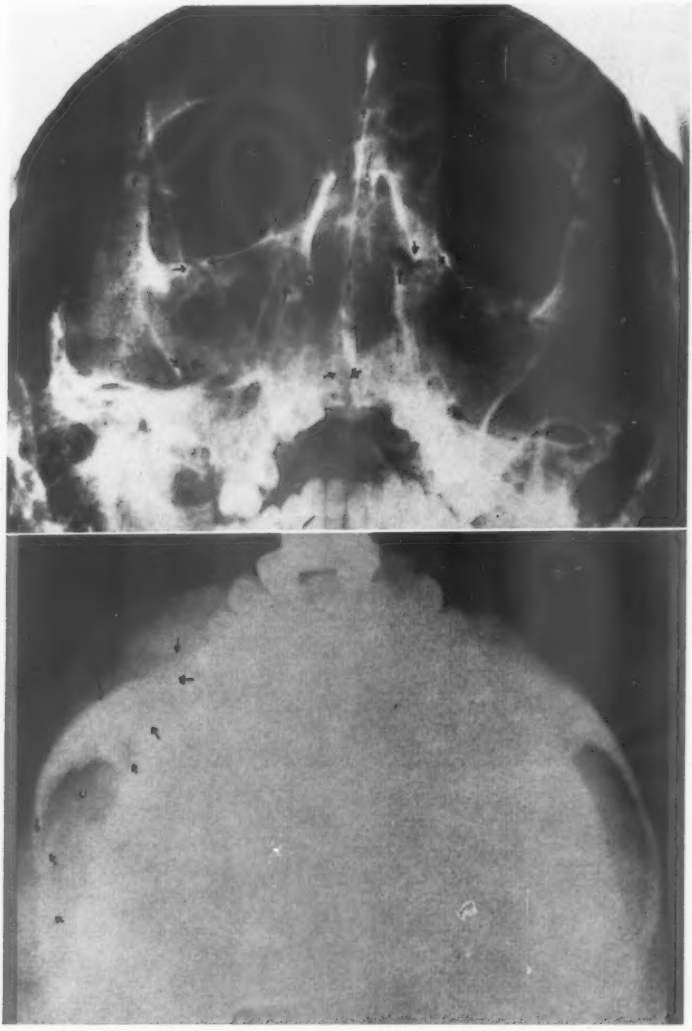

Fig. 19.

Fig. 18.—Case 3. Waters' position view showing severely injured patient with complete maxillary fracture through the nose, the palate, and the orbit.

Fig. 19.—Case 3. Mental vertex showing severely compressed malar bone and zygomatic arch.

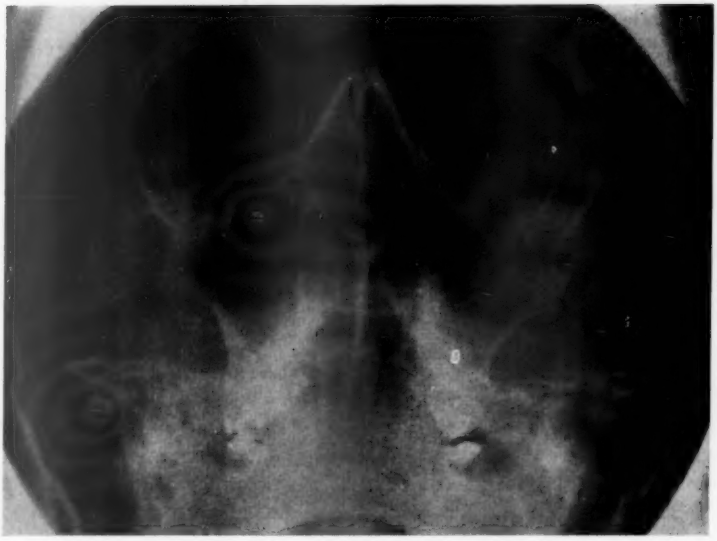

Fig. 20.—Case 3. Postoperative Waters' position view.

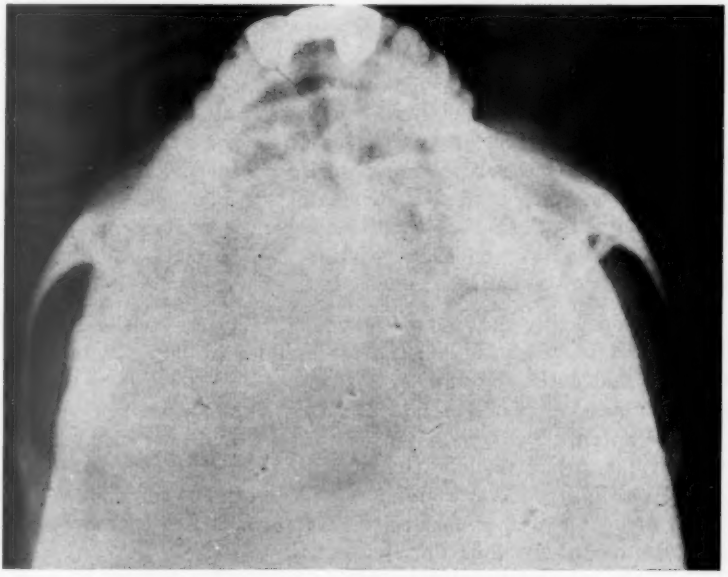

Fig. 21.—Case 3. Postoperative submental view.

of Bell's palsy. On the other hand, should severe tissue laceration accompany the impact, then injury to the various branches of the facial nerve may result in permanent damage to the motor distribution of the seventh nerve. This is not uncommon in severely injured patients who have middle facial and malar fractures. It is wise to examine all such patients for neurological injury prior to surgery and to clearly point out the injury; otherwise, the patient may think that this is a result of surgery.

SURGICAL PROCEDURES

A number of surgical procedures have been used in the treatment of these fractures. They are as varied as a simple lifting with a towel clip to an open reduction through the antrum. There is no one definite procedure for all types of fracture; hence, no specific one is advocated. In some instances, however, one procedure may have certain advantages over another.

A simple fracture of the zygomatic arch may require no more than judicious grasping with a towel clip for elevation. This same fracture may be approached through the mouth by making a small incision beneath the arch and elevating the fracture with an appropriate instrument. On the other hand, Gillies' approach through an incision in the temporal fossa can be used to pass an instrument beneath the depressed arch and thus accomplish elevation.

Some operators feel that the external approach is to be preferred in all cases. Others prefer the intraoral approach. Each has its merit and place.

OPEN REDUCTION OF ORBITAL RIM

When wide separation of the frontomalar suture occurs, it may not be possible to align the fragments properly unless the site is approached directly. A small skin incision over the area, followed by blunt dissection, will expose the area in a simple manner. A hole is drilled through each side of the fracture, and a wire suture is passed. This will serve to hold the fragments in proper position while the remainder of the injury is treated.

ANTRAL APPROACH TO MALAR FRACTURE

If we consider only the case of the malar fracture which is depressed into the antrum but in which there is no horizontal maxillary fracture, then the procedure is that outlined below.

The intraoral approach to the maxillary sinus should not be attempted through a semilunar incision as in the Caldwell-Luc operation. It is to be remembered that when the maxillary wall is severely comminuted there is always the possibility that the loss of bone beneath the incision will be great enough to prevent a proper closure. Oroantral openings caused by this type of procedure are usually difficult to close.

The incision should be made on the crest of the ridge in the edentulous patient, or around the cervical portion of the teeth when they are present, and a wide flap elevated (Fig. 11, *A*). The edges of the flap should be placed

over an area of bone which is not fractured. After the flap has been elevated, the fracture site comes into view. With care, it is possible to elevate one of the fractured pieces of the bony wall with the periosteal attachment (Fig. 11, *B*). It is not necessary to be heroic in excising bone to examine the sinus fully. It is only necessary to remove blood and any obvious debris present. Once this has been accomplished, obvious antral pathology should be removed and the malar fragment elevated with one of the many instruments that may be used for this procedure.

In cases of three to five days' duration, it is necessary to use only minimum force. One hand should be kept outside the face, on the infraorbital region, while the other exerts intra-antral pressure. When gross discrepancies have been corrected, an opening into the sinus is made beneath the inferior concha in the inferior nasal meatus (Fig. 11, *C*). A hemostat is passed through this opening from the nose into the sinus. The edge of an oiled tape dressing is picked up with this instrument and pulled out into the nose. This is left attached to the hemostat while the antrum is being packed. Next, the opposite end of the dressing (approximately 5 yards) is placed in the most distal position of the antrum and folded in layers against the fractured walls. Enough of the dressing is packed into the antrum to maintain the reduction of the fractured parts (Fig. 11, *D*). This is checked repeatedly by the fingers of the left hand placed on the outside of the face. It is not remiss to overreduce the fractured parts, for there is a tendency for the area to collapse slightly while healing takes place. In any case, enough of the dressing is placed to maintain the reduction and the remainder is cut off. The flap is repositioned and sutured with 000 silk. The finger should then be used, intraorally, to mold the buccal maxillary wall to some semblance of normal. The orbital rim should be reduced to a point where no elevation is discernible in the fracture site. The lateral orbital rim also should be replaced in the same manner. If the zygomatic arch had been depressed, it should have been elevated by passing an instrument beneath the soft tissue and palpating the reduction. When the oral closure has been completed, the excess nasal dressing is cut off with the hemostat and a small portion is left hanging from the nares. This is used to remove the packing when that procedure becomes necessary seven to ten days later.

Since these patients are usually operated upon under general anesthesia, it is necessary to provide some means of preventing the patients from compressing the reduction while reacting to the anesthetic. It is also necessary to prevent them from rolling onto the operated side in their sleep. This is accomplished with a pot-cover type of handle made from tongue blades and adhesive tape. The ends are padded with felt and placed over the temple and down onto the mandible. This is retained with a Barton bandage, and the patient is allowed to wear the apparatus for several days (Fig. 14). Once the danger of restlessness is past, the apparatus is removed and the patient is instructed to sleep elevated on several pillows. On the tenth day the end of the dressing is picked up with a hemostat and pulled gently from the nasal aperture.

The procedure may be made comfortable for the patient by an adequate premedicative dose of Demerol. The patient will be more comfortable if we maintain the antibiotic coverage for several more days after removal of the dressing.

COMPLICATIONS

It is not wise to attempt to move a fracture of the malar bone after a two- or three-week period. Such a fracture is so firmly fixed by this time that moving it can become a major procedure. When other injuries make it impossible to reduce such a fracture properly, the resultant deformity is more easily corrected by inserting a cartilage or bone graft over the malar bone and filling out the deformity. This is a much less drastic procedure than refracturing the bone. In cases in which diplopia is a result of this condition, it may be necessary for the ophthalmologist to insert a cartilage graft beneath the eye in the floor of the orbit so as to raise the eye to the same level as the uninjured eye. This usually corrects the visual difficulty caused by the difference in the height of the eyes.

In some cases the transorbital wiring procedure is required when the frontomalar junction is too widely separated to permit proximation by any other method. This also may be necessary in some instances of wide separation of the orbital rim in the region of the infraorbital canal. I have never had to resort to transorbital wiring in this area, but others have reported finding it necessary to use this procedure.

CONCLUSIONS

Some of the problems associated with the treatment, diagnosis, and aftercare of fractures of the malar bone and zygomatic arch have been discussed. Some suggested treatment procedures have been covered.

1931 Mott Ave.

TROPICAL OSTEOMYELITIS OF THE JAWS

Report of a Case Involving the Whole Body of the Mandible

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OSTEOMYELITIS of the jaws is frequently encountered in tropical areas of the East in a very severe form which is almost never seen in Europe and America. It is not clear whether the acute destructive course and rapid spread of the disease are occasioned by combinations of circumstances of undernourishment, neglect, overcrowding, and bad climatic conditions or whether Eastern peoples, particularly Chinese, have a racial susceptibility toward the disease. The picture is so different, however, from that seen in the West, that one feels justified in referring to osteomyelitis of the jaws in the East as a tropical disease.[1, 2, 4, 6, 8, 9]

Members of the Chinese race may develop osteomyelitis with astonishing rapidity. The exaggerated condition which frequently develops is what I have called the "subacute spreading stage" of the disease. This stage may develop immediately from an infection of dental origin or from a fractured jaw, or it may represent a lighting-up of a chronic osteomyelitis as a result of a lowered state of general health or the presence of acute infection elsewhere in the mouth, nasopharynx, or antrum. The disease spreads most commonly in the mandible through the spongiosa and causes a breakdown of the buccal and labial walls of the bone, the lingual walls being the last to be affected. A spread upward to the ascending rami and downward to the inferior borders of the angles of the mandible is also common. Multiple suppurating fistulas appear early at the gingival margins and in the labial and buccal mucosa. The teeth in the affected areas become loose, there is increased fetor of breath, and the patient experiences malaise with a moderate rise in temperature.

Although most of the cases of tropical osteomyelitis of the jaws that I have encountered in Hong Kong have been in poor, undernourished persons, a few patients have not been in that category, and the rapid spread of the disease in such persons does seem to indicate a racial susceptibility. There was the case, which I reported in 1953,[3] of a well-nourished and well-built 24-year-old male Chinese farmer who developed osteomyelitis from pericoronitis

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with a history of six months. This was associated with the third mandibular molars, which were in good position. The dentition was perfect, with no missing teeth, no caries, and no restorations, and what obviously had been (judging by the upper jaw) an excellent gingival condition. Osteomyelitis suddenly developed at both third molar regions and spread within a few days to meet in the midline, leading to the loss of all lower teeth and the entire mandibular alveolar process.

The following case is presented as being interesting, first of all, because of the advanced stage of destruction of the mandible and, second, as a study of the bony healing process.

CASE REPORT

The patient, a 39-year-old Chinese woman, was a boatwoman who lived and worked exclusively on a sampan in the harbors and off the coasts of Hong Kong and south China. She was referred to me by the general surgeon of a public hospital which she had attended for treatment of her jaw and of multiple sinuses discharging pus on her face and neck (Fig. 1).

A. *B.*

Fig. 1.—Patient when first seen, showing asymmetry of face and discharging facial sinuses.

History.—One year previously she had experienced a toothache with alveolar abscess. She received no treatment, and eventually the abscess subsided. Pain and swelling recurred one month later, but the only treatment on this occasion had been the application of plasters (counter-irritant?) to her face. The swelling progressively increased and spread to the other side with trismus, loosening of lower teeth, and discharge of pus on the face and from inside the mouth. General and familial history was noncontributory.

Examination.—The patient was a Chinese woman of average height and medium build with the bodily toughness of an outdoor worker. Her face was grossly swollen on both sides, and several sinuses on the neck and cheeks were discharging pus (Fig. 1). She was in a lowered state of health but had been living a normal life aboard her sampan and had walked

without assistance up a steep hill from the bus to the dental clinic. On examination of her mouth, it was found that all teeth were present except the first and third molars on both sides. With the exception of a carious third molar, all maxillary teeth were in good condition. Of the mandibular teeth, only the second molars were carious, but all the teeth were loose, with pus running from the gingival margins and from numerous sinuses in the buccal and labial mucosa. A bony sequestrum 2 to 3 inches in length was exposed in the buccal sulcus. The occlusion was gagged in the molar region, resulting in open-bite to the extent of about ¾ inch in the incisor region (Fig. 2). This was caused by a pathologic fracture on both sides of the mandible. In fact, the body of the mandible consisted of several separated fragments which could be palpated from inside the mouth. Roentgenograms (Fig. 3) showed what appeared to be complete necrosis of the body and inferior parts of the ascending rami of the mandible with multiple pathologic fractures. A diagnosis of chronic osteomyelitis of the mandible was made. The patient was hospitalized on the same day, and penicillin therapy was instituted. Her temperature on admission was 102° F., but it rapidly fell to 100 degrees and later to 99.5 degrees after a few days of bed rest. Physical examination revealed nothing significant aside from the local condition and the attendant weakness from infection and lack of proper diet caused by difficulties in mastication. X-ray examination of the lungs showed nothing abnormal except old scars. The Kahn test was negative. Culture from pus revealed only staphylococcal pathogens. Nourishing soft food and vitamins were prescribed, and bed rest for three weeks was imposed to improve the patient's physical condition before operation. In the meantime, an acrylic upper cap splint was constructed with short vertical posts on the occlusal surface. This splint was designed to fit into and hold in place the "gutter" splint which would be constructed to replace temporarily the necrosed bone to be removed at operation (Fig. 4).

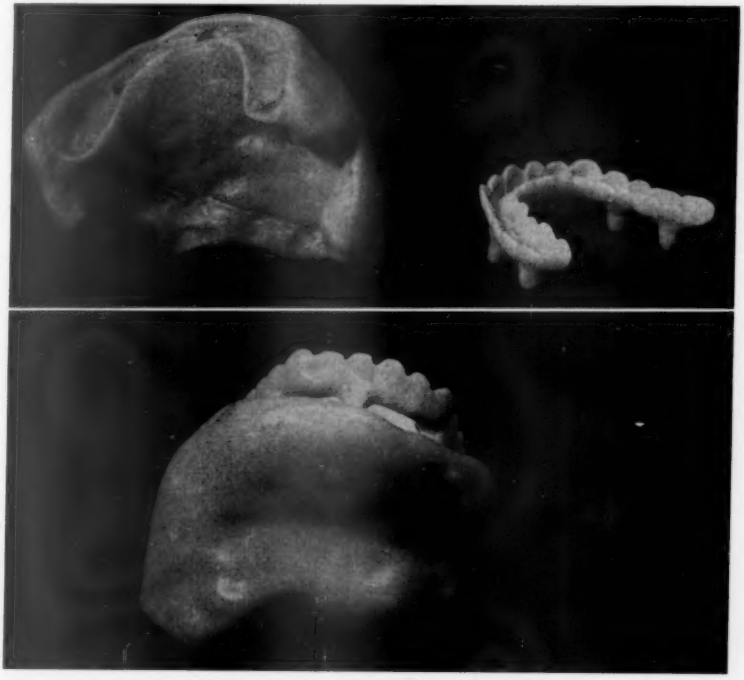
Fig. 2.—Patient when first seen, showing the bite gagged open because of multiple pathologic fractures of the mandible.

Operation.—On Oct. 10, 1949, under intratracheal general anesthesia, the lower teeth were extracted and large flaps were reflected buccally, labially, and lingually to expose the infected bone. Anterior border incisions were also made high up both ascending rami to the coronoid processes. Detached and infected bone was removed in about eight major pieces. About half of the right ascending ramus in its inferior aspect was trimmed away with rongeurs, and approximately two-thirds of the left ascending ramus was also removed. The only attached and viable bone in the main body of the mandible was a triangular area lingually in the midline carrying the geniohyoid tubercles. It was thought that if this one piece of bone were left, it also would be likely to become infected and prejudice the healing of the rest of the area. Accordingly, this section of bone was dissected out, the attachments of the geniohyoglossus and geniohyoid muscles being severed.

Fig. 3.—Radiographs of patient on first admission to hospital, showing necrosis and pathologic fractures of the mandible.

The lower jaw was then in the condition of a deep gutter of soft tissue with no ossified tissue whatever from a point two-thirds of the way up the left ascending ramus around to halfway up the right ascending ramus. An impression of this gutter was taken in Kerr's impression compound. Because of the large mass required and the softness of the tissues into which it had to be placed, the impression was taken in three parts and was extended to fit around the vertical post extensions of the acrylic cap splint previously made for the upper teeth. The impression was cooled and removed from the mouth. The gutter area of the mandible was then lined with petrolatum gauze, and Stent's composition was molded into place and fitted against the upper cap splint. When the patient had regained consciousness, a chin bandage was applied to keep the mold in place fairly firmly. No drain was inserted, as all infected bone had been removed and there was no longer any possibility of deep infection. Hemorrhage had been brisk at one stage of the operation, but the blood loss was not so great as to constitute a danger, and no blood transfusion was necessary.

A.



B.

Fig. 4.—*A*, Acrylic cap splint with vertical extensions and acrylic "gutter" splint to maintain the shape of the soft tissues after sequestrectomy. *B*, Cap splint fitted into "gutter" splint.

The day after the operation the patient was comfortable, with little pain, and complained only of difficulty in swallowing. The temperature was 98.8° F. Four days after the operation an attempt was made to fit an acrylic gutter splint made from the impression taken at operation, but unfortunately the tissues had shrunk somewhat and the splint could not be fitted down. The temporary stent was therefore built up with wax in the mouth, the petrolatum gauze having been removed; it was processed in opaque acrylic and finally fitted four

days later (Figs. 4, 5, and 6). The bite was deliberately gagged open to apply positive pressure to the gutter splint by means of a jaw bandage. This caused some discomfort to the patient in that dribbling of saliva occurred incessantly.

The patient wore the splint night and day for the next two months, remaining in the hospital on a soft diet, but she then began to complain of pain located in the right ascending ramus. The temperature was elevated. The splint was eased without relief, and it became obvious that there was infection in that area. Seventy days after the first operation, under intratracheal anesthesia, the right ascending ramus was opened; a bony sequestrum about 1 inch square was discovered and removed, and the bone end was nibbled back to sound bone. The splints were not placed back into the mouth, as the lower splint no longer fitted comfortably. The second sequestrectomy was adequate; no more pain was experienced, and the temperature returned to normal. At this time there was a definite feeling of firmness in the mandibular area on both sides. The patient was discharged from the hospital Jan. 12, 1950, eighty-four days after the first operation.

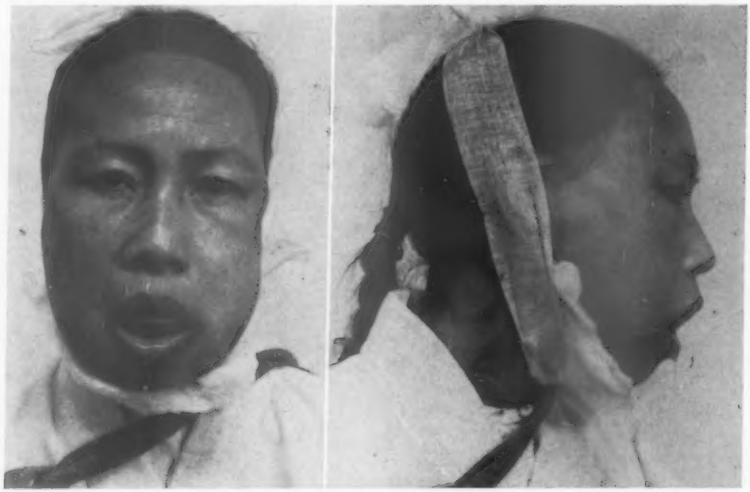


Fig. 5.—Upper and lower splints in position (jaw bandage has been slipped back to show facial contour) eight days after sequestrectomy.

She returned to her boat and was not seen again until Feb. 10, 1950. All swelling had disappeared, there was no pain, and she was fit and well. The alveolar regions were firm as far anteriorly as the canine areas on both sides. Roentgenograms showed calcification from the ascending rami to the first molar areas (Fig. 6).

One month later, on March 13, 1950, clinically there appeared to be calcification on the left side almost to the midline, but on the right side it did not seem to have progressed anteriorly beyond the canine region.

The patient was seen monthly until the end of May, 1950, when it was decided that a lower prosthesis should be fitted. Three maxillary teeth that were biting down on the lower alveolus were extracted. A lower denture of acrylic was fitted eight months after the original operation. This prosthesis was not a good fit because of the complete lack of alveolar ridge, but it was considered desirable for two reasons: (1) it improved the patient's appearance and made her more acceptable to her friends and relatives and (2) it was hoped that it would stimulate bone growth and strengthen muscular action. Four months later this denture was remodelled, since there was a high bite on the left side,

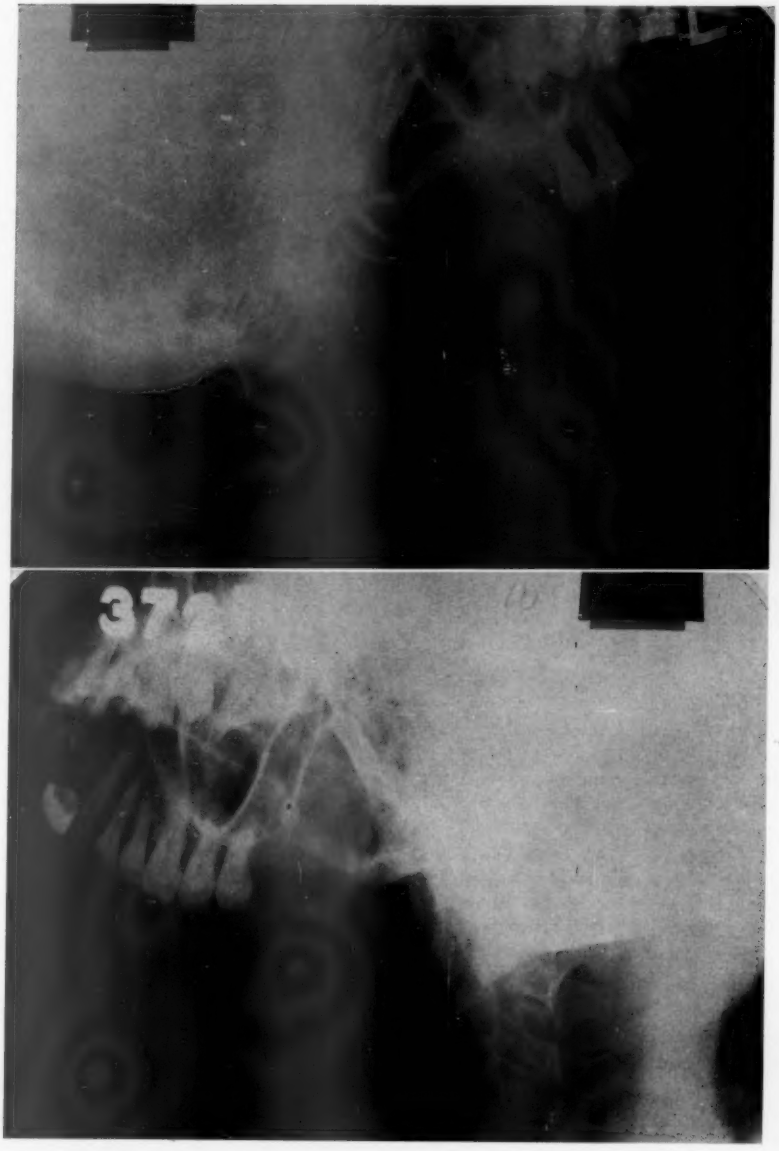


Fig. 6.—Radiographs showing early calcification of the mandible 113 days after sequestrectomy.

probably due to increased bone formation (Fig. 7). At this time (November, 1950) it was observed clinically and radiographically that calcification had continued beyond the midline from the left side and was approaching the right canine region anterior to which calcification on that side apparently had not proceeded.

The patient was seen at about two-month intervals, and by June, 1951, it was obvious that the approaching "arms" of calcification of the new mandible from left and right had met at the right canine region but were not uniting. The only union was apparently of a fibrous nature and was firmer than six months previously.

A. *B.*

Fig. 7.—*A*, Profile of patient eleven months after operation. *B*, Profile of patient while wearing prosthesis.

The patient was then seen at six-month intervals. In November, 1952, she was experiencing considerable difficulty in retaining her lower denture, and it was felt that little improvement could be obtained with a new prosthesis unless the diminutive alveolar ridges could be made more prominent. It was recommended to the patient that an attempt be made to form buccal sulci by placing epithelial inlays.

Eventually in February, 1954, the patient decided to submit to this operation. She was admitted to the hospital where an upper cap splint and a lower block splint, both of acrylic, were constructed.

Operation.—On Feb. 17, 1954, under intratracheal anesthesia, an incision was made in the buccolabial aspect of the alveolar process and widened and undercut as much as possible. Skin was taken from the inner aspect of the right thigh and applied to the fresh area in the mouth by means of a stent of Kerr's impression compound held firmly under the lower block splint which was maintained in firm contact with the upper cap splint by means of a jaw bandage. It was considered that the mandible was too small and irregular to be suitable for the placing of circumferential wires to hold the lower splint in place. After the patient had been returned to the ward and had recovered consciousness, firm traction was applied by means of a leather strap under the jaw, attached by a pulley to a one-pound weight. Nasal feeding was instituted but was replaced after two days by liquid feeding from a feeding cup with a long rubber tube extension. The patient was instructed to swallow as much of the rubber tube as possible before sucking in in her food. In this way, contamination of the graft was kept to a minimum without subjecting the patient to the discomfort of

prolonged nasal feeding. Progress was satisfactory, and on March 1, 1954, the splints were removed from the mouth and cleaned, and the mouth was syringed. The epithelial inlay had taken well. Impressions were then made in Kerr's compound, and the splints were replaced. Bite and try-in were checked on successive days and on March 5, 1954, a solid acrylic prosthesis was inserted to maintain the shape of the graft area. This was articulating against the upper teeth in a slightly open-bite position. After a few days the patient was discharged from the hospital, and she again returned to her boat and her family.

Two months later a full lower acrylic denture was constructed which, although not very stable was a great improvement over what formerly had been possible (Fig. 8). Her appearance was considerably improved, although mastication presented some problems. Roentgenograms taken in February, 1955, showed the state of calcification of the new diminutive mandible. There has been no obvious change in the condition since that date.

Fig. 8.—The patient in February, 1955, five years and four months after original operation (compare with Fig. 1, *A* and *B*).

This patient was last seen on Sept. 12, 1956. There was still a state of non-union at the right mandibular canine region, but a moderately powerful bite was possible with the full lower denture made thirty months earlier. This denture was loose, but the patient seemed very happy with it and would not hear of any remodelling or replacements. She was fit and well at the age of 46. Since her first operation in 1949 she had given birth to three additional children, her twelfth, thirteenth, and fourteenth!

DISCUSSION

This case illustrates two important points: (1) the periosteum's great powers of resistance to infection and its subsequent ability to lay down large areas of new bone, even without a residual bony matrix, and (2) the inhibition of bone formation in the presence of infection. No signs of new bone were found at the operation, although the osteomyelitis had been in the chronic or subacute stage for at least ten months. Less than four months after the operation a large amount of new bone had been laid down.

Calcification on the right side did not occur anterior to the canine region, whereas on the left side calcification proceeded progressively to the incisor region and then across the midline to the other side. The advancing areas of calcification met at the right canine region, but unfortunately they did not join forces and unite. The delay in calcification on the right side was apparently due to the residual infection of the right ascending ramus which made a second sequestrectomy in that area necessary. This had upset the wearing of the acrylic gutter splint which, because of pain, had been left out of the mouth for several days before the second sequestrectomy. Because of the degree of contraction of the soft tissues, which had occurred very rapidly while the splint had been left out, it was decided not to make a new splint. Nevertheless, it is believed that the wearing of the gutter splint for two months had done much to maintain the shape of the facial contour which would have been much worse without support in the stages of initial healing.

In spite of the gross loss of mandible and the corresponding muscle attachments, after the first week following operation the patient appeared to suffer no undue embarrassment of speech. According to Hopkin and McEwen,[5] Robinson[7] considers the genioglossus muscle "to be the only structure capable of exercising the quick and exact control of the tongue necessary for making the movements in the anterior part of the mouth where 80 per cent of consonantal sounds are made." However, dissection of this muscle and removal of its bony attachment did not long prevent adequate tongue control for speech.

SUMMARY

1. So-called "tropical osteomyelitis of the jaws" is discussed.

2. A case of tropical osteomyelitis of the mandible with multiple pathologic fractures is described.

3. A diminutive body of mandible was formed and calcified following complete removal of the infected body and parts of the ascending rami.

4. A full lower prosthesis was fitted and worn eight months after operation.

5. After the first prosthesis had been worn for three years and eight months, the buccal and labial sulci of the new small mandible were deepened and epithelial inlays were placed so that an improved lower prosthesis could be fitted.

6. When last seen, the full lower prosthesis had been worn for sixteen months with reasonable comfort and masticatory ability.

The thanks of the author are due Dr. G. Graham-Cumming, M.B., Ch.B.(Edin.) D.T.M.(Liv.), M.R.San.I., D.P.H.(Edin.), formerly Acting Director of Medical and Health Services, Hong Kong, for permission to use departmental records and to publish this paper.

The case report was included in shortened form in the author's dissertation in respect to Regulation 7 for the diploma in public dentistry, University of St. Andrews, June, 1952.

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MALIGNANT MELANOMA

Report of a Case

Harry M. Seldin, D.D.S., S. Daniel Seldin, D.D.S., William Rakower, D.D.S., and William Jarrett, D.D.S., New York, N. Y.

MALIGNANT melanoma, or the shortened form melanoma, is one of the most malignant and dangerous forms of neoplasm. A more common lesion than is usually suspected, it comprises about 1 to 2 per cent of all malignant tumors and about 20 per cent of all skin tumors. Melanomas may occur anywhere in the body and are found in the eye, at the junction of the skin and mucous membrane, and at the site of the body orifices.[1, 2, 3]

A review of the literature, however, reveals that primary malignant melanomas of the mouth, as distinguished from those elsewhere in the body, are extremely rare. According to Greene and colleagues,[6] only sixty-two cases of primary malignant melanoma of the mouth have been reported. The term *melanoma* was introduced by Carswell in 1838, and the first oral case reported was one described by Weber in 1859 which involved the mucosa of the palate.[4, 5]

Many of the cases reported in the older literature were diagnosed on the basis of clinical evidence alone. The criteria for ascertaining whether the oral malignant melanoma actually arises from the oral mucous membrane should be based on (1) demonstration of malignant melanoma in the oral mucosa, (2) the presence of intraepidermal (junctional) activity, and (3) inability to demonstrate any other primary site.[6]

Most intraoral malignant melanomas occur on the mucosa of the maxilla. However, cases have been reported on the tongue, cheek, floor of the mouth, and parotid gland.[5, 7-9] Melanomas, like carcinomas, are more common in men than in women.

The melanoma kills not because of its local disturbance, but rather because of early metastasis. The tumor grows rapidly, infiltrates the underlying bone, and causes metastasis to the regional lymph nodes, spreading to the cervical and axillary chains.[10, 11] From these nodes the tumor may further metastasize by a hematogenous route, with extension to the lungs, brain, and other organs of the body.[12]

SYMPTOMS AND CLINICAL SIGNS

The chief complaint is usually of a painless, pigmented, intraoral swelling. There may be associated tender lymph nodes.

The lesion is usually soft, and it may appear nodular, flat, or papillary. The color varies from a bluish hue to dark brown to jet black. Often its dark nodular or warty surface gives it a "blackberry" appearance.[4-6, 11, 13]

Ulceration may occur with ensuing secondary infection. In more advanced cases, infiltration of bone causes the teeth to become loose. As the melanoma increases in size and spreads, it may cause interference with speech and swallowing, and trismus. Generalized metastasis follows, and death ensues.

HISTOLOGY

Thoma[11] reports: "The intraoral melanoma does not vary greatly from the malignant mole in its histologic make-up. The cells which differentiate into melanoblasts vary greatly in shape and arrangement, depending on the origin of the tumor. They may occur as spindle cells or round cells, large or small, and different cells may be found in the same tumor. The stroma is generally very delicate and scarce. At other times there is enough connective tissue to divide the tumor into segments. The secondary growth, lymph node involvement or metastasis to the internal organs, may be similar to the primary lesion, but sometimes the metastatic growth is not of a melanotic nature."

Baxter[5] states: "Pigmentation is marked in most cases, but is unevenly distributed so that in one part of the tumor the cells may be filled with pigment and in another they may contain none. If the pigment cells become overloaded, they may break down and liberate the melanin which is absorbed by phagocytic histocytes . . . the degree of pigmentation has no relation to the malignancy of the tumor."

TREATMENT AND PROGNOSIS

As in all cases of malignancy, the earlier a diagnosis is made, the better are the chances of successful treatment. Treatment will also be influenced by whether the lesion is primary or metastatic. If we discover a metastatic oral melanoma, we can assume that probably the tumor has at the same time invaded the large organs of the body as well. The prognosis in such cases is extremely grave, and treatment is palliative and conservative by means of radiation.[11] Death usually occurs within one to three years after treatment.[15]

The treatment of choice for the primary oral lesion in most cases is wide surgical excision and dissection of the cervical lymph nodes which drain the involved area. Radiation has been used, but it is not considered the treatment of choice in most cases.[16]

CASE REPORT

The patient, an 83-year-old nonambulatory (wheel chair) man, was first seen in the office, accompanied by his physician, on Nov. 5, 1956. He complained of a bulging dark mass on the gingiva and palate. He had first noticed the mass about three or four months prior to this visit and at that time consulted his dentist (Fig. 1). At that time the dentist advised the patient to seek further consultation. More than three months passed, however, before this advice was acted upon. During this time the lesion had grown in size.

The patient was senile, with a previous history of coronary thrombosis and apoplexy. Several years prior to this visit he had a papillary carcinoma of the urinary bladder, which was treated surgically. He was aged, disabled, and bedridden.

Fig. 1.—Photograph taken by the patient's dentist in August, 1956, shows the pigmented areas on the labial gingiva with diffuse extension upward in the left cuspid area.

Fig. 2.—Photograph taken on Nov. 5, 1956, shows bulging black palatal mass and numerous flat pigmented areas. The inset in the lower left-hand corner is the biopsy specimen.

Upon examination, a firm, bulging, darkly pigmented mass could readily be seen involving the left anterior third of the hard palate. The mass was essentially jet black and had a roughened surface. Adjacent to the mass and extending to the right side of the palate were numerous flat, pigmented areas, varying in color from brownish to black.

The palatal mass went around the premolar to the labial gingiva about the upper anterior teeth, and extended in a diffuse manner up the mucobuccal sulcus. There was also evidence of pigmentation labially in the upper right anterior region (Figs. 2 and 3). The upper anterior teeth adjacent to the mass were mobile. No cervical lymphadenopathy was noted. The clinical picture was typically malignant melanoma.

Roentgenograms revealed a marked loss of alveolar supporting bone. The palatal bone appeared intact (Fig. 4).

Under local anesthesia, a portion of the palatal mass was removed electrosurgically for pathologic examination. The pathologist's* report follows:

"*Macroscopic description:* The specimen is a flat piece of mucous membrane, 2 by 1.5 cm. and 1 cm. in depth. The submucous tissues are irregularly discolored with a black-brown pigment.

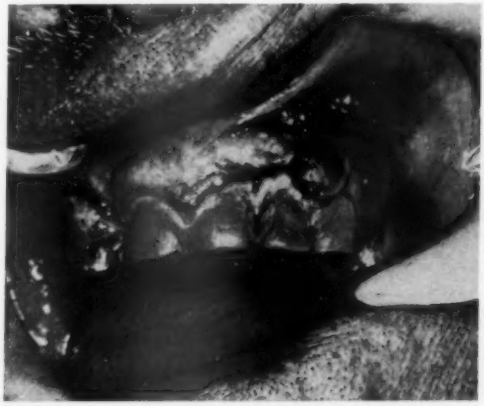

Fig. 3.—Photograph taken on Nov. 5, 1956, shows the extension of the pigmented area labially and upward into the buccal sulcus.

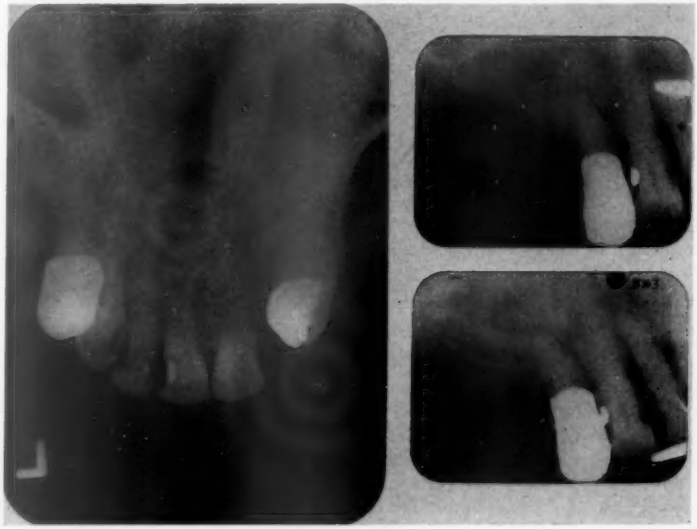

Fig. 4.—Roentgenograms taken on Nov. 5, 1956, show the degree of alveolar bone absorption. The palatal bone appears to be intact.

*Charles Darlington, M.D., New York, N. Y.

"*Microscopic description:* Only residuals of the squamous roof remain. Most of the surface cells have sloughed and a bank of fibrin and entangled inflammatory cells now face on the oral cavity. Deep to this slough an unbalanced, unrestrained growth of anaplastic pigment-bearing cells provides bulk to this specimen. The growth pattern is roughly the plexiform solid cord. These cords are outlined by slender strands of connective tissue. The transformed cells are frequently spindle shaped. Nuclear chromatin is gathered into coarse chunks and heavily chromatinized. Brown melanin pigment is obviously an elaborated product of these cells, for many of them are loaded with it. This pigment is even found in the supporting connective tissue, implying perhaps phagocytosis by other pigment-carrying cells (Fig. 5).

"*Pathologic diagnosis:* Malignant melanoma."

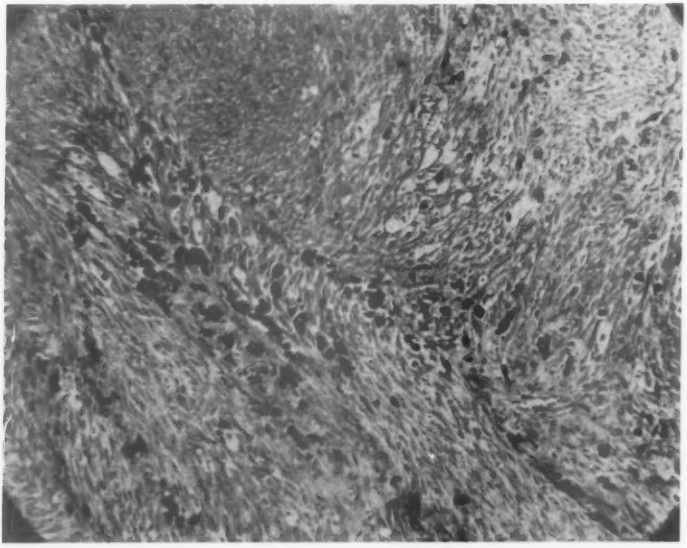

Fig. 5.—Unrestrained anaplastic pigment-bearing cells are seen, with supporting strands of connective tissue.

Follow-up.—On Nov. 9, 1956, the patient was seen by the Pack Medical Group and was thereafter cared for by them. A summary from the records of their treatment and the patient's course follows.

Because of the patient's age, senility, history of coronary thrombosis, and apoplexy, surgery as a method of treatment was eliminated. Instead, it was decided to treat the lesion with radiation.

An acrylic splint was constructed to cover the entire palate and labial mucosa, to contain the radium used for therapy. It was constructed in two sections, for ease of insertion, the sections joined by means of wire hooks and eyelets. The splint contained a lead filter to protect the patient's lips, cheeks, and tongue from radiation. Depressions were made in the superior surface of the splint for the positioning of the platinum radium tubes, each containing 50 mg. of radium salt. Once these tubes were placed, the depressions were covered with a paraffin covering so as not to change the contour of this surface* (Fig. 6).

*The prosthesis was constructed by A. Ackerman, D.D.S., New York, N. Y.

On Nov. 18, 1956, this splint was inserted for four hours with 550 mg. of radium. The total dosage was about 2,100 mg. hours of radium therapy.

By Dec. 4, 1956, an excellent regression of the lesion was noted.

By Feb. 7, 1957, the excellent regression had been maintained and the radiation reaction had almost completely subsided.

On April, 13, 1957, a tender node in the right carotid group was noted. The mouth was better.

On May 25, 1957, the mouth was doing well, but the node in the right carotid group was larger and hard, and it was thought that it undoubtedly contained metastatic melanoma. Again, because of the patient's age and debility, surgery was deferred. Instead, on June 11, eight gold radon seeds were inserted into the enlarged node for a total of 1,650 millicurie hours.

On July 13 the mouth lesion was seen to be still well controlled, but the patient was coughing and expectorating blood and appeared anemic. Roentgenograms showed unquestionable evidence of miliary metastasis to both lungs.

On July 22, Thio-TEPA, 10 mg. intramuscularly, was started and was to be continued weekly for four weeks, after which the patient was to be re-evaluated.

On August 20, however, the patient died as a result of generalized metastasis.

Fig. 6.—Acrylic lead-lined splint with grooves to contain the platinum radium tubes.

DISCUSSION

Although malignant melanomas have a characteristic appearance, other pigmented lesions of the mucous membrane may simulate them. Some of the heavy metals may cause a bluish black or black discoloration of the gingiva and buccal mucous membrane. It was not uncommon some years ago, when bismuth was used in the treatment of lues, to see the so-called bismuth line about the cervical gingiva or buccal mucous membrane.

We occasionally see bluish black or black discolorations of the mucous membrane as a result of the accidental tattooing of the mucosa with amalgam or carborundum from a disk.

Pigmentation of the oral mucosa is seen also in Addison's disease, and may be associated with intestinal polyposis. In the darker-skinned persons diffuse brownish discoloration of the gingiva is a normal characteristic.[14]

Usually, however, the blackness of the melanoma, tumor formation, and the history of rapid growth delineate it from other oral pigmented areas. Biopsy, of course, removes all doubt.

In the case presented here, the lesion apparently did not grow with alarming rapidity. The somewhat restrained growth of the lesion in this aged and senile patient is probably a reflection of the slowing up of all his metabolic processes.

It is noteworthy that, despite the regression of the primary oral lesion following radiation, a metastatic cervical node was noticed five months after the start of treatment and death ensued four months after the node appeared. About fourteen months had elapsed from the time the lesion was first noticed by the patient until his death.

SUMMARY AND CONCLUSION

1. The characteristics of oral malignant melanomas are described.

2. A case of primary oral malignant melanoma is presented.

3. The role of the dentist in the recognition of these lesions and his function as part of the team which treats it are illustrated.

4. The importance of early recognition is stressed.

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57 W. 57TH ST.

EXODONTIA

VIPER VENOM FOR CONTROL OF POSTEXTRACTION HEMORRHAGE

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INTRODUCTION

PROLONGED hemorrhage after dental extractions is a complication often encountered in oral surgery. Postoperative bleeding from an extraction wound may be expected to last for a period of five to fifteen minutes. Hemorrhage exceeding this time will generally require special attention. A steady flow of blood from single or multiple sockets is usually controlled with oxidized cellulose (Oxycel gauze*) which has been moistened with viper venom (Stypven*). There are instances in which more elaborate techniques may be indicated to cope with an unusual situation; however, the method described here has been clinically effective in a sufficient number of cases to merit consideration.

MATERIAL

Stypven is supplied in a package containing two vials. One vial contains 0.5 mg. Stypven in dry form. The other vial contains 5.0 c.c. of sterile distilled water to which has been added 0.5 per cent phenol to act as a preservative. The contents of these vials, when mixed, make a 1:10,000 solution of Stypven used as a laboratory reagent for the detection of abnormalities in the clotting mechanism of the blood[1-2] or as an effective hemostatic agent.[3] If stored in a refrigerated, well-stoppered bottle with precautions to avoid gross contamination,[4] it is stable[5] for a period of several months. A 5.0 c.c. syringe and 20 gauge needle are used for mixing the vials and for application to the Oxycel gauze at the time of use.

METHOD

The following procedure has been found successful in the management of many cases of postextraction hemorrhage requiring special attention:

1. Inject the area with a local anesthetic to minimize discomfort.

*Oxycel gauze is manufactured by Parke, Davis & Company, Detroit, Michigan; Stypven by Burroughs Wellcome & Co., Tuckahoe, New York.

2. Remove all tissue debris and clotted blood from the extraction wound.

3. Moisten small pieces of Oxycel gauze with Stypven and place loosely in the socket.

4. When indicated, suture the area to eliminate bleeding from the margins of the wound.

DISCUSSION

Stypven is similar to thromboplastin and converts prothrombin to thrombin in the presence of calcium. Favorable results can be expected only when the venom comes in direct contact with the bleeding area. It is therefore essential that all clotted blood and tissue debris be removed from the socket before the moistened cellulose is applied. This absorbable dressing is not removed from the socket. If the dressing and clot are lost, a sedative dressing may be required to keep saliva and debris from causing discomfort until granulation occurs. Stypven has the unusual property of arresting the bleeding without the formed clot losing its bright, fresh appearance. This characteristic may lead the unfamiliar operator to conclude that bleeding has not stopped, and he may erroneously abandon this treatment in favor of another method.

SUMMARY

Viper venom is an effective hemostatic agent that converts prothrombin to thrombin in the presence of calcium. When applied to Oxycel gauze and placed in the socket of a recently extracted tooth, it will solve the vast majority of prolonged bleeding problems encountered in the practice of oral surgery.

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CUTICULAR CALCULUS ATTACHMENT

El. G. Voreadis, D.D.S., and H. A. Zander, M.S., D.D.S., Rochester, N. Y.

THE attachment of calculus to the tooth root has been described by Zander.[1] According to his findings, it is attached in four ways: (1) the organic matrix of calculus is attached to the secondary cuticle; (2) the cuticle is absent and the calculus matrix is attached to minute irregularities in the cementum surface; (3) organisms have penetrated into the cementum and are continuous with the organisms in the calculus matrix, and (4) calculus is located in areas of cementum resorption.

This investigation deals with the attachment of calculus to the enamel surface, particularly with the cuticular type of attachment. Cuticular substances have been described in the past and have been distinguished as "primary cuticle" (a product of ameloblasts) and as "secondary cuticle" (a horny structure formed by the squamous cells of the epithelial attachment).[2]

Waerhaug[3] believes that primary and secondary cuticle are of the same nature and that it is possible for them to be formed on a nontooth surface in twenty-three days. He supported his statement by microphotographs.

On the other hand, Manly[4] described a structureless "pellicle" which is different from the other dental accumulations and Nasmyth's membrane. It is similar in quality to the enamel cuticle, and it is formed anew on the tooth surface each time that one removes it. This pellicle is a keratin-like material and is characterized by its freedom from structure and microorganisms.

Vallotton,[5] from his work on the same pellicle, concluded: "The keratin-like behavior of the pellicle . . . could be accounted for by the chemical changes of mucin rather than by a chemical identity of pellicle and keratin . . . formation of keratin on teeth without specific cellular activity appears impossible from a biological standpoint."

The observation by Zander that a cuticular attachment of calculus exists has since been verified by Mandel and Levy,[6] who observed a cuticle-like material which separated the calculus mass from cementum and dentine. Waerhaug[7]

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and Shroff[8] also described a cuticle between the calculus and the enamel. All these authors assumed that this cuticular structure which they observed was the "secondary" or "primary" cuticle—in other words, a cuticle that was laid on the tooth surface prior to the formation of the calculus substance. Mandel and associates,[9] however, observed a cuticle on celluloid strips between the celluloid material and bacterial plaques which had formed on the surface of the celluloid. This immediately raised the question as to the nature and origin of this cuticular material, since in this case it could not have been formed by epithelial cells. Yet they (Mandel and colleagues) state that it resembles the cuticle over the surface of the enamel and cementum which had been described by other investigators. They further state: "In view of our findings the problem of calculus attachment should be more fully investigated."

The purpose of this investigation is to shed further light on this problem of cuticular calculus attachment.

STATEMENT OF PROBLEM

We would like to answer two questions:

(1) Is the cuticle that formed on the celluloid strips identical with the cuticle that forms on the enamel surface? In other words, after all cuticles are removed from the enamel surface will a similar cuticle form without the presence of epithelium in contact with the enamel surface?

(2) What is the incidence of this cuticular type of attachment of calculus on enamel?

MATERIAL AND METHOD

In order to compare the cuticle forms on film strips with that which forms on the enamel, we selected a patient who had six remaining lower anterior teeth which needed extraction. We removed all calculus on these six teeth and polished them thoroughly with pumice. Previous studies had shown that such treatment will remove any cuticular structures that might be present on the enamel. With orthodontic wire, we then fastened Mylar film, 0.001 inch thick, to three of the teeth. These strips remained on the teeth in the mouth for sixty-two days. At the end of this experimental period the strips were removed from these three teeth, and the three teeth that had not been covered by strips were extracted. All six specimens were fixed in formalin and prepared for celloidin embedding. Serial sections were made of these specimens and stained with hematoxylin and eosin. Similar sections were stained with stains[10] said to be specific for keratinous substances (sodium plumbate and picric acid). The controls for these stains were sections of human eponychium and rat skin containing hairs.

In order to study the incidence of a cuticle between calculus and enamel, we observed under the microscope forty specimens of calculus on enamel surface. Twenty-four were teeth from human beings, some of which had had regular dental prophylaxes, and sixteen were autopsy specimens which were furnished to

us for this study by Dr. S. S. Arnim. The latter were prepared by the double-embedding method which retains the enamel matrix in the sections. Ten of the latter specimens were also stained for bacteria.

RESULTS

The calculus on the three teeth and on the three Mylar strips was found to be attached in the same way, through a cuticle-like substance which, with hematoxylin and eosin staining, appeared dark pink and well distinguished from the calculus mass, the tooth, and the Mylar strip (Fig. 1). With picric acid staining, all these cuticles appeared bright yellow (Figs. 2 and 3). The keratinized structures of the hair, skin, and eponychium appeared various tones of yellow to dark blue (Fig. 4).

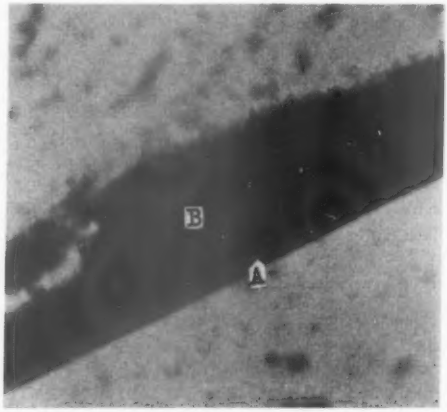

Fig. 1.—Photomicrograph of cuticle (*A*) and calculus (*B*) which have formed on Mylar strip after sixty-two days in mouth. (Hematoxylin and eosin stain. Magnification, ×220.)

With the sodium plumbate staining, all the cuticles appeared brown-yellow, while hair, skin, and eponychium varied from black to brown-yellow, probably because of variable sulfur content of the different tissues. However, the comparison of the different stained sections supported the idea that the cuticle-like substance to which we found the calculus attached may be a keratin-like substance.

In thirty-eight of the forty specimens examined, a cuticular attachment of calculus to the enamel could be found. In some the cuticle covered the major portion of the enamel, while in others it was much more limited in extent. As was seen on the root surface, however, we noted other types of calculus attachment to the enamel. Bacteria were particularly prominent, and their attachment on enamel irregularities, cracks, and lamellae was seen (Fig. 5).

DISCUSSION

From these results, it is obvious that there can form on tooth surfaces, as well as on Mylar strips, a cuticle that, for all practical purposes, looks the same as

Fig. 2.—Cuticle (*A*) and calculus (*B*) stained with picric acid. This specimen is from a tooth of the same patient as those in Figs. 1 and 3. (Magnification, ×900.)

Fig. 3.—Picric acid stain of cuticle (*A*) and calculus (*B*) from Mylar strip of tooth adjacent to the one in Fig. 2.

Fig. 4.—Eponychium. The highly keratinized part (*A*) appears yellow with picric acid stain. (Magnification, ×220.)

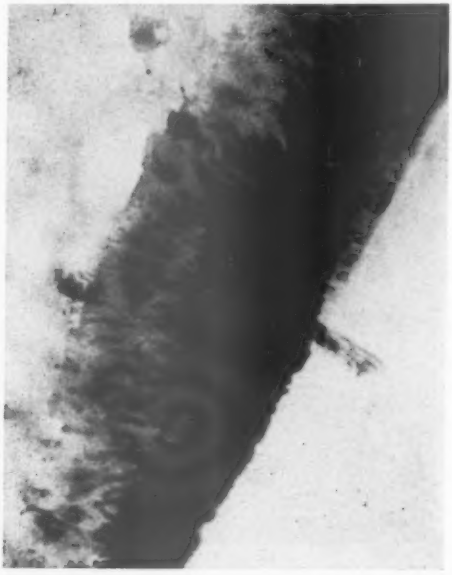

Fig. 5.—Bacterial stain of calculus attachment to enamel. Bacteria can be seen in lamella. (Human autopsy specimen courtesy of Dr. S. S. Arnim.)

what is described in the literature as the "dental cuticle" formed by epithelium. This finding sheds some light on the dilemma in which Manly and Vallotton found themselves when they described and investigated a "pellicle" as distinct from the dental cuticle. Yet, they had to admit that, based upon chemical tests, the pellicle was closely related to keratin. Manly had no explanation for this chemical similarity, and Vallotton thought that the keratin-like behavior of the pellicle was due to chemical changes of mucin since he just could not rationalize that keratin could appear on teeth without specific cellular activity (epithelium).

Since one often finds epithelial cells in various degrees of degeneration in the saliva and in the calculus mass, we can make the hypothesis that desquamated epithelial cells play a part in the formation of the cuticle. That seems more likely than the hypothesis that this material is a product of bacteria. This needs further investigation.

The incidence of cuticular attachment to the enamel in this series was much higher than that previously described for the root surface. The fact that bacterial attachment was less prominent on the enamel than has been shown to be the case on the cementum was probably the result of the enamel's providing fewer irregularities in which the bacteria could grow. Therefore, the cuticular method of attachment was more suitable for the attachment on the enamel surface.

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ORAL MEDICINE

THE DENTAL ASPECTS OF INFANTILE CORTICAL HYPEROSTOSIS

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INFANTILE cortical hyperostosis is a recently recognized clinical entity. A report by Roske[11] probably was the first dealing with a case of it, but the syndrome was not fully recognized until the clinical and roentgenologic features were presented in 1945 and 1946.[4, 6, 17]

A disease of obscure cause and pathogenesis, infantile cortical hyperostosis affects infants less than 5 months old.[5, 15]

Usually it is characterized by the sudden onset of soft-tissue swelling of the face, thorax, or extremities, together with hyperirritability, fever in a fluctuating course, and roentgenologic evidence of periosteal formation of new bone underlying the soft-tissue swellings.[5] In most cases the disease regresses spontaneously after a few months, and ordinarily the hyperostoses are invisible twelve months after the soft-tissue swellings and fever have subsided. Bowman and co-workers,[1] however, reported that they had seen one patient with persistent mandibular enlargement at the age of 3 years, and Caffey reported that one of his patients exhibited mandibular asymmetry at 4 years 9 months of age.

The unvarying presence of mandibular involvement is one of the most striking features of the syndrome; indeed, so constant has been this finding that it is doubtful whether a diagnosis of the disease should be made in its absence.[5] The mandibular changes in almost all cases are the reason that this disease process should be of extreme interest to the dental profession. The study reported here was undertaken to determine the effects of infantile cortical hyperostosis on the teeth and jaws of young children who had had the disease in infancy.

Abridgment of thesis submitted by Dr. Burbank to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Dentistry.

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MATERIALS AND METHODS, WITH FINDINGS

The subjects used in this study were eleven children seen in infancy at the Mayo Clinic and determined there to have had infantile cortical hyperostosis.

This diagnosis had not been reached immediately, and in several cases it had not been the first recorded. The final decision had been reached eventually on the basis of the patient's age and the roentgenographic, clinical, and laboratory studies.

Previous Findings.—Four of the eleven children were girls and seven were boys. The age of onset ranged from 3 weeks to 4 months (Table I).

TABLE I. SIGNS AND SYMPTOMS AT ONSET IN ELEVEN PATIENTS SUFFERING FROM INFANTILE CORTICAL HYPEROSTOSIS

CASE	SEX	AGE AT ONSET (MONTHS)	CHIEF COMPLAINT	FEVER	IRRITABILITY	BRAWNY EDEMA OF FACE
1	F	4	Swelling of right mandible		+	+
2	F	4	Tenderness of jaws and chin	+	+	
3	M	2	Swelling of cheeks, squint of left eye	+	+	+
4	F	2½	"Moon-shaped" face	+	+	+
5	F	1	Swelling of jaws	+	+	+
6	M	2	Swelling of right scapula			
7	M	2	Tenderness of left arm		+	
8	M	1	Swelling of face	+		+
9	M	¾	Swelling of face and right arm		+	+
10	M	2½	Swelling of right cheek	+	+	+
11	M	2½	Swelling of jaw and eyelids	+	+	+

The chief presenting complaint in eight of the eleven patients had been swelling of the face, cheeks, and jaws (Fig. 1). The remaining three patients had had initial complaints as follows: tenderness of jaws and chin to touch, swelling of the right scapula, and tenderness of the left arm (Table I).

TABLE II. SKELETAL INVOLVEMENT IN ELEVEN PATIENTS SUFFERING FROM INFANTILE CORTICAL HYPEROSTOSIS

CASE	MANDIBLE	RIBS	SCAPULA	CLAVICLE	HUMERUS	ULNA
1	+					
2	+				+	
3	+					
4	+	+				
5	+					
6	+	+	+	+	+	+
7	+		+	+		
8	+					
9	+					
10	+	+				
11	+					
Totals	11	3	2	2	2	1

Fever had been present in seven of the patients. Irritability had been a feature in nine cases. A brawny, nonpitting edema of the face had been evidenced in eight (Table I).

Fig. 1.—Posteroanterior roentgenogram showing massive soft-tissue swelling overlying the affected bone in a patient at the age of 4 months.

Fig. 2.—Lateral-jaw roentgenogram showing the subperiosteal proliferation of new bone. Note the laminations of new bone formation and the roughened surface contour of the inferior border of the mandible.

In all eleven cases of infantile cortical hyperostosis included in this study, the mandible had been affected by the subperiosteal proliferation of new bone (Table II and Fig. 2). In six of the eleven cases, the mandible had been the only bone involved. The ribs had been involved in three, the humeri, scapulae, and clavicles in two, and the ulna in one.

Laboratory blood studies had been made in eight cases. Three patients had had anemia, with the erythrocyte count varying from 2.96 million to 3.78 million per cubic millimeter of blood. The lowest value for hemoglobin in this group had been 8.5 grams per cubic centimeter of blood, and the highest 10.1 grams. The leukocyte counts in these eight patients had ranged from 6,300 to 24,400 per cubic millimeter. Five of the eight had had counts in excess of 14,600. In the five cases in which a sedimentation rate had been obtained, four had been abnormally high, varying from 60 to 120 mm. per hour by the Westergren method.

An immediate impression of infantile cortical hyperostosis had been obtained in four of the eleven cases. One case had been diagnosed on the basis of a biopsy report. In the remaining six cases a total of ten provisional diagnoses had been considered before the correct diagnosis of infantile cortical hyperostosis had been made. Four of these six patients had been thought to have mumps or parotitis (Fig. 1).

Present Study.—Recall letters were sent to the parents of the eleven children recorded as having had infantile cortical hyperostosis, asking them to bring the children back to the Mayo Clinic for clinical and roentgenographic examination with specific reference to the teeth and jaws. Of these eleven requests, six were fulfilled.

Patients Studied Clinically and Roentgenographically.—The ages of the six children in this group ranged from 1 year 11 months to 7 years 3 months, and the time lapse from onset of disease to the time of this study ranged from one year eight and one-half months to six years eleven months. All of these children were studied both clinically and roentgenographically.

The clinical dental examinations were thorough. The teeth were examined for size and shape and for quality of tooth enamel. The number of teeth erupted was noted and correlated with the normal eruption time and sequence.

The occlusion of the teeth with their antagonists in the opposite jaw was noted. In the two instances of malocclusion encountered, impressions of the teeth and dental arches were obtained with an alginate dental impression material, and casts were made from these impressions (Figs. 3 and 4). The size, shape, and occlusion of the teeth and jaws were studied from the casts.

Jaw roentgenograms, including lateral and posteroanterior views, were obtained in each case; and intraoral dental roentgenograms were obtained from four children old enough to cooperate. All of these were studied with special reference to the gross size and symmetry of the mandible. Residual deformity was sought in the views of the cortex, and the roentgenologic quality and relative size of the spongiosa were noted.

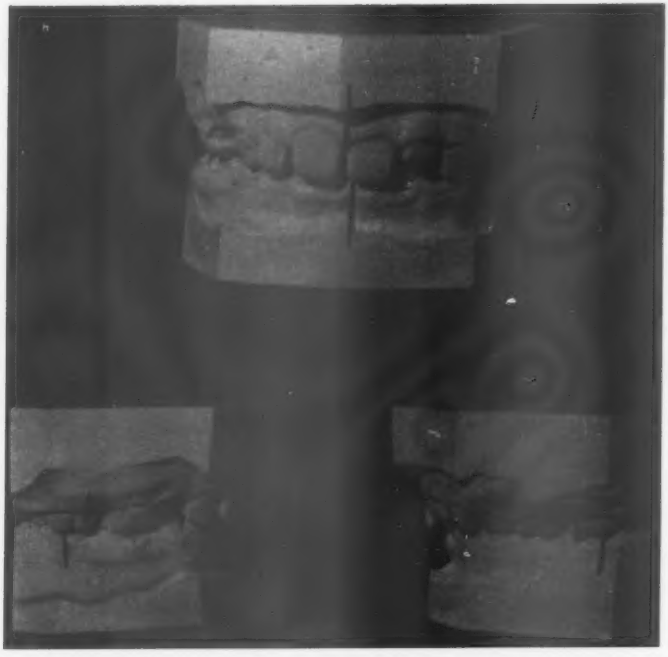

Fig. 3.—A cast demonstrating an Angle Class II, Division 1 malocclusion. The mandible is retruded in relation to the maxilla by 4 mm.

Fig. 4.—A cast demonstrating an "open-bite" malocclusion.

Two of the six children who returned for study had severe malocclusion. One had an Angle Class II, Division 1 malocclusion, in which the entire mandible was in a retruded relationship to the maxilla by the amount of 4 mm. (Fig. 3). The second malocclusion was an Angle Class I. The anteroposterior relationship of the mandible to the maxilla was normal but, due to premature contact of the deciduous molar teeth, the anterior teeth were in an "open-bite" relationship (Fig. 4).

Among the six cases in this group, evidence of residual deformity of the mandible was found in the roentgenograms of four (Table III); among these, clinical evidence of malocclusion was seen in two (Fig. 5), and the other two were marked by slight residual thickening of the cortical bone.

TABLE III. LATE CLINICAL AND ROENTGENOGRAPHIC JAW FINDINGS IN SIX PATIENTS WHO SUFFERED FROM INFANTILE HYPEROSTOSIS

CASE	QUALITY OF MANDIBULAR BONE		RESIDUAL DEFORMITY OF MANDIBLE	MALOCCLUSION
	CORTICAL	MEDULLARY		
1	Normal	Normal	Slight residual asymmetry; left body slightly larger than right in vertical dimension	Mandible in retruded relationship with respect to maxilla; Angle Class II, Division 1 malocclusion
2	Normal	Normal	None	None
4	Normal	Normal	Slight residual asymmetry; midline of the mandible left of midline of maxilla	Increased vertical dimension in body of mandible on both sides caused premature contact of molars and open bite of incisors
5	Normal	Normal	None	None
9	Slight residual thickening	Normal	Very slight residual cortical thickening	None
11	Slight residual thickening	Normal	Minimal asymmetry near angles and ascending rami	None

Patients Studied by Roentgenogram Only.—The remaining five children of the original eleven did not return to the clinic for this study. Among this group the ages at the time of the study ranged from 2 years 1 month to 4 years 6 months, and the interval from onset of the disease to the time of the study ranged from one year ten months to four years four months.

The parents of these children were requested, through their local physicians, to obtain roentgenograms of their children's teeth and jaws. These roentgenograms were studied, as were those of the children who revisited the clinic, for residual deformities of the mandible. In addition, among the five, abnormalities with respect to the dentition had to be determined solely from roentgenographic study, since these patients were not available for actual clinical study.

One child of this group who did not return appeared entirely normal roentgenographically (Table IV). Four were found to have residual deformity of the mandible. One of these had a very slight malocclusion caused by a shifting of the midline of the mandible to the right (Fig. 6). Only one presented any abnormality of the cortical bone, this being in the nature of persistent lamination and slight thickening of the cortex.

Fig. 5.—Varying degrees of residual mandibular asymmetry eight months (*a*), one year four months (*b*), and six years eleven months (*c*) after onset of infantile cortical hyperostosis. Note the shift of the midline of the mandible to the right.

TABLE IV. LATE ROENTGENOGRAPHIC JAW FINDINGS IN FIVE PATIENTS*

CASE	QUALITY OF MANDIBULAR BONE		RESIDUAL DEFORMITY OF MANDIBLE	MALOCCLUSION
	CORTICAL	MEDULLARY		
3	Normal	Normal	No residual deformity	None
6	Normal	Normal	Definite asymmetry at angles and ascending rami; right angle appeared thicker than left	Midline of the mandible displaced to right of midline of maxilla
7	Normal	Normal	Very slight asymmetry; right coronoid process bigger than left	None
8	Normal	Normal	Definite asymmetry at angles and ascending rami	None
10	Slightly thickened and laminated	Normal	Right ascending ramus shortened, creating asymmetry; right body larger in vertical dimension than left	None

*No follow-up clinical dental examination was performed.

Common Findings.—None of the eleven patients showed roentgenographic evidence of abnormal change in the quality of the enamel. The number of teeth and the eruptive sequence of the dentition were within normal limits in all cases. There was no roentgenographic evidence of abnormal medullary bone or of malignant change in the jaws.

Fig. 6.—Roentgenogram of a patient three years seven months after onset of infantile cortical hyperostosis, showing gross asymmetry of the mandible at the angles with marked displacement of the mandible to the right. Note the difference in size and density at the angles of the jaw.

COMMENT

Roentgenographic evaluation is a valid means of detecting the presence of dental malformations and this method of study is supported by many dental investigators.[8, 12, 13] Sarnat and co-workers[13] have reported on the value of

roentgenograms of unerupted permanent teeth as an aid in the early diagnosis of dental malformations. Logan and Kronfeld,[8] in a study of the human jaws and surrounding structures from birth to the age of 15 years, used roentgenographic examinations extensively.

During the period of development the dentition and skeletal structures are affected in their growth and mineralization by poor general health and by many specific diseases.[21]

Fever and Enamel Hypoplasia.—One of the characteristic features of infantile cortical hyperostosis is intermittent fever,[6, 14, 16] and many dental investigators[2, 7, 10, 21] attribute a causal role in the production of enamel hypoplasia to systemic diseases, especially those that are febrile in their nature.

Enamel hypoplasia is a dental manifestation of constitutional disease which affects the enamel increments of deciduous or permanent teeth that are in the process of formation during the disease period and which remains as a record of the time of occurrence and duration of the disease. This record is comparable to that of arrested growth in long bones, and according to Sarnat and Schour,[12] it can be studied by roentgenographic, clinical, and histologic methods. These workers carried out a chronologic analysis of enamel hypoplasia in relation to systemic disease and showed that two-thirds of the instances of enamel hypoplasia occurred during the period from birth to the end of the first year.

Our own study suggests that the effect of infantile cortical hyperostosis on the teeth with regard to enamel hypoplasia is negligible. In roentgenographic evaluation of the teeth of the eleven patients in this study, specific attention was focused on the enamel, but in no case was there any roentgenographic finding of enamel hypoplasia.

Eruptive Sequence and Infantile Cortical Hyperostosis.—At the start of this study it was thought that infantile cortical hyperostosis might, in addition to having a possible effect on the enamel of the teeth, affect the eruptive sequence of teeth. Boyle,[2] in his discussion of the eruption of teeth, stated that a bony scar on the occlusal side of a permanent tooth germ might retard eruption. It was conceivable that the cortical hyperostosis might leave such a bony scar and, in so doing, inhibit the normal eruption of the teeth.

Massler and Schour,[9] in their studies of tooth development, discussed theories of eruption thoroughly. After extended research into the problem, they believed that the eruptive force, or the force responsible for tooth movement during eruption, might be related to the vascularity of the tissue which surrounds the tooth. Whatever the ultimate source of the eruptive force, clinical and experimental evidence shows that it probably is active in the periapical region of the tooth.

Sidbury and Sidbury[14] suggested that infantile cortical hyperostosis is the clinical manifestation of an inherited defect of the arterioles supplying the affected areas. The resulting hypoxia causes a focal necrosis of the overlying soft tissues and a proliferative reaction of the periosteum.

The results of our study, however, indicate that infantile cortical hyperostosis probably has no effect on the eruptive sequence of the teeth.

Anemia and Mandibular Bone.—One of the findings of the laboratory blood studies in patients with infantile cortical hyperostosis is anemia, as evidenced by both a low erythrocyte count and a low hemoglobin concentration. Thoma,[21] in his discussion of general diseases affecting the jaws, pointed out that anemia in infancy may produce pathologic changes in the mandible in the form of a thinning-out of the cortices and a wide trabeculation with greatly increased radiolucency of the bone.

The results of this present study show that no untoward effects in the mandible can be attributed to the anemia present during the acute phase of infantile cortical hyperostosis.

Constitutional Disease and Malocclusion.—Strang[19] emphasized the effect of constitutional diseases in childhood as an influential factor in the production of malocclusion. Probably the most important constitutional upset of childhood in respect to influence upon the production of malocclusion is that caused by allergy,[19] and Bowman and co-workers[1] believed infantile cortical hyperostosis to be the result of an allergic phenomenon.

Nevertheless, the two patients in our study who presented malocclusion had no history or evidence of any allergic reactions.

Development of Tooth and Arch Relationships.—As in the permanent dentition, there is a key to normal tooth and arch relationships in the deciduous dentition. This is found in the locking of the cusps at the second molars.[19] It will be recalled that the second deciduous molars, both maxillary and mandibular, are but miniatures of the first permanent molars in their respective arches. In normal occlusion the mesiobuccal cusps of each maxillary second deciduous molar occlude between the mesiobuccal and distobuccal cusps of the mandibular second deciduous molars, and the mesiolingual cusps of the maxillary second deciduous molars lie in the central fossae, surrounded by all of the cusps (usually five in number) of the mandibular deciduous molars.

Two instances of severe malocclusion have been reported in this study. The first described showed a retruded position of the mandible with respect to the maxilla (Fig. 3). Forward growth of the body of the mandible is associated with the preparation of sufficient room to accommodate the permanent molars in their positions behind the deciduous dentition. This occurs by additions of bone on the posterior border of the rami and absorptions of bone along the anterior borders of these processes. By this double process, the body of the bone is lengthened and the molars are released from their primary positions within the rami and given freedom to erupt. Surface additions to the alveolar process in the incisor area also add to the forward growth of the mandible.

Broadbent[3] has pointed out that there seems to be a site of active growth at the angle of the mandible. It is interesting to note that in our study this was the area most commonly affected by the residual deformity in the nature of mandibular asymmetry (Fig. 6).

In the second instance of malocclusion described, the premature contact of the posterior teeth which caused the open-bite relationship of the anterior

teeth was itself due to an increase in the vertical dimension in the body of the mandible.[18]

Strang[19] stated that increase in vertical height of the mandible is due to the eruption of the teeth and the appositional growth of the alveolar process to maintain and support these dental units in function. The entire lower third of the face actually is wedged apart when these erupting mandibular teeth meet the downward-moving maxillary teeth.

Thoma[20] made an extensive study of the principal factors controlling the development of the mandible and the maxilla. He found that the development of the mandible seems to be influenced principally by muscular function and its associated increase in collateral blood circulation and that because the most powerful muscles are attached to the ramus the growth of the body of the mandible occurs principally in the posterior part.

Biopsy evidence indicates that the pathologic process of infantile cortical hyperostosis involves the overlying soft tissues as well as the bone and periosteum,[14] and it has been suggested already[14] that infantile cortical hyperostosis is the clinical manifestation of an inherited defect of the arterioles supplying the affected areas, with the resulting hypoxia causing focal necrosis of the overlying soft tissues and musculature with a proliferative reaction of the periosteum. The combination of this finding and hypothesis with Thoma's suggestion[20] (that the growth of the mandible seems to be influenced principally by muscular function and its associated increase in collateral blood circulation) may provide an explanation for the underdevelopment or retrusion of the mandible in the first malocclusion discussed. Furthermore, this same chain of thought may explain the mandibular asymmetry so frequently found at the angles of the mandibles in the patients studied in this series (Fig. 6).

SUMMARY AND CONCLUSIONS

Eleven patients who had infantile cortical hyperostosis were studied for the purpose of determining the effect of this disease upon the teeth and jaws. There were four girls and seven boys in the series, and their ages at onset of the disease ranged from 3 weeks to 4 months. The ages at the time of this study ranged from 1 year 11 months to 7 years 3 months. The interval from onset to the time of this study varied from one year eight and one-half months to six years eleven months.

The most common chief complaint at onset of the disease among the patients in this series had been swelling of the face and jaws. Fever, irritability, and brawny edema of the face had been frequent accompanying complaints. Laboratory studies had shown the presence of anemia in three of the patients.

In six of the eleven cases, the mandible had been the only bone involved. Other bones involved were the ribs, scapula, clavicle, humerus, and ulna.

The clinical diagnosis of infantile cortical hyperostosis had been made immediately in only four of the eleven cases. In the remaining seven cases there had been ten provisional diagnoses, with mumps or parotitis the most common.

For this study, six patients were re-examined clinically and roentgenographically and five patients were evaluated on the basis of new roentgenographic evidence only.

Eight of the eleven cases studied had roentgenographic evidence of a residual asymmetric deformity of the mandible, the most common site being the angle and ramus of the mandible.

Two of these eight patients also exhibited severe malocclusion of the teeth.

We conclude that the effect of infantile cortical hyperostosis upon the teeth is negligible. Even despite the febrile component of the syndrome, no enamel hypoplasia has been observed under the conditions of this study.

Also, the evidence indicates that the mandible is affected by the disease to the extent that residual asymmetry of the mandible may result, with concomitant malocclusion.

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HEREDITARY HEMORRHAGIC TELANGIECTASIA INVOLVING THE ORAL CAVITY

Review With Report of a Case

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A SEARCH of the dental periodicals of the past forty-five years reveals only one reference to hereditary hemorrhagic telangiectasia and a case report of this disease. Yet, here is a disease which invariably has extensive oral manifestations. A proper understanding of the disease is essential if periodontal, operative, prosthodontic, or oral surgical procedures are to be undertaken. Furthermore, its resemblance at times to hemorrhagic purpura (and, indeed, to all purpuras) presents a challenge.

HISTORY

Hereditary hemorrhagic telangiectasia was first described in the medical literature by Sutton[1] in 1864, Rendu[2] in 1896, Osler[3] in 1901, and Weber[4] in 1907. Osler, Weber, and Rendu, in that order, are the men usually associated with the disease, and often it is referred to as Osler-Weber-Rendu disease.

DESCRIPTION AND ETIOLOGY

This disease is a vascular anomaly characterized clinically by bleeding or hemorrhage and anatomically by dilatations of capillaries and venules anywhere in the skin and mucous membranes. This vascular abnormality seems to be transmitted as a simple Mendelian dominant in both sexes. The sexes are equally affected. The telangiectases may be found in childhood, but they increase in number as age advances and bleeding may not commence until adulthood is reached. Some persons rarely or never suffer from hemorrhage; this may explain the apparent skipping of members of a whole generation, which should not occur if the disease is transmitted as a true dominant. Occasionally, perhaps, a mutation occurs or lesions are all internal, thereby seeming to skip some persons.

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SYMPTOMATOLOGY

Symptoms of the disease are bleeding and hemorrhage with resulting anemia. Epistaxis is common, but bleeding may come from telangiectases in any location of the mucous membrane or skin. Cases of bleeding from the mucous membrane of the tongue, the buccal mucosa, and the gastrointestinal,[5-7] respiratory, or genitourinary tract have been reported. The bleeding from the telangiectases may be spontaneous or it may follow slight trauma.

GROSS APPEARANCE

The multiple telangiectases may be of pin-point size or larger, sometimes even reaching the size of a pea, and they often become spiderlike, especially in elderly patients. Spider telangiectases particularly appear in the mucous membranes of the nose, the oral cavity, and the skin of the face and neck, usually after the age of 40 years. They increase in size and number with age. This is exactly the age at which the patient to be reported here developed his first lesion.

Fig. 1.—Hereditary hemorrhagic telangiectasia. The multiple telangiectases on the tongue vary in size from the size of a pin point to the size of a pea. The walls of the blood vessels consist of a thin layer of endothelium or dilated capillaries or venules. Trauma to the angiomas may cause severe bleeding.

The lesions consisting of multiple telangiectases may be seen by the dentist on any mucous membrane of the oral cavity and on the face, hands, lips, ears, conjunctivae, and finger tips. The patient may or may not have pallor, according to the blood loss at the time. The color of the lesions is due entirely to the blood within the dilated vessels (Figs. 1, 2, and 3).

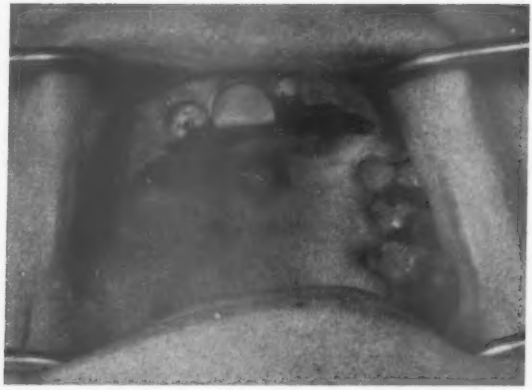

Fig. 2.—Multiple telangiectases are also seen on the hard palate. Note the variation in size. The small telangiectases resemble purpuric spots from which differential diagnosis must be made.

Fig. 3.—The characteristic telangiectases are also found on the tips of the fingers.

BLOOD FINDINGS

The blood findings are those which result from blood loss, and a diagnosis of anemia will be made if hemorrhage has occurred. Bleeding time, coagulation time, clot retraction time, and platelet count are within normal limits. Bleeding is not from a blood constituent deficiency or an abnormality in the blood but from a spontaneous blood vessel rupture.

TELANGIECTASIA AND PURPURA

Differential diagnosis from the purpuras is important. When the telangiectases are small, they may resemble purpuric spots, but hereditary hemorrhagic telangiectasia usually has the following triad which purpura does not have: (1) habitual hemorrhage, (2) multiple telangiectases, and (3) familial history. A simple clinical test to differentiate may be made by producing pressure with a glass slide through which the color of the lesion may be seen. In hemorrhagic telangiectasia, the lesion will fade partially on pressure, whereas hemorrhagic purpuric spots do not fade when pressure is applied. Furthermore, as stated previously, there are no blood test abnormalities in hereditary familial telangiectasia, but in purpura the bleeding time is prolonged.

HISTOPATHOLOGY OF TELANGIECTASIA

Histologic study shows the walls of the involved vessels to be extremely thin and consisting merely of a layer of endothelium. This is not characteristic of all vascular lesions, however, for there may be a dilation of the capillaries and venules with no deficiency of elastic tissue. In the upper corium large sinusoidal spaces can usually be seen just under the epidermis.[8]

CASE REPORT

F. S. K., a 69-year-old white man, was admitted to the Veterans Administration Hospital in New York City on March 18, 1957. He was born in the United States. He served in the Navy in World War I and had worked as a federal employee until his retirement.

His chief complaints on admission were shortness of breath, swelling of the legs, chest pains, and blood loss. His admission diagnosis was congestive heart failure and hereditary hemorrhagic telangiectasia. His temperature was 99.2° F. and his blood pressure was 160/78.

The heart symptoms were of approximately five years' duration and had gotten progressively worse. He had been admitted for treatment of congestive heart failure and anemia at various other hospitals.

Previous History.—The patient had a history of frequent nosebleeds, which first started during his childhood. To the age of 18 he had nosebleeds about every other month, and they stopped spontaneously. While he was in the Navy in World War I and between 1919 and 1930 there were very few nosebleeds. About 1930 he began having nosebleeds again; he noticed angiomas (bright red spots) on the left middle finger and red spider-like lesions on the lower lid of the left eye. In 1936 the patient sought medical advice because of the increased number of nosebleeds. There was no increase in angiomas at that time. From 1936 to 1950 the patient's nose was cauterized several times for nosebleeds with doubtful success. In 1950 Adrenalin nose plugs were used for hemorrhages, since cauterization did not stop the bleeding when the patient had a severe hemorrhage. At about this time, there was an increase in angiomas of face, hands, and chest.

In 1950 the patient was hospitalized for epistaxis and myocarditis. The nasal membranes were sclerosed and the bleeding stopped. The patient was severely anemic at that time, with a red cell count of 2,000,000. He was put on vitamin K, iron, and liver injections and therapeutic multivitamin therapy. Bleeding from the nose never resumed during the hospital stay and has not recurred to date. He had several transfusions for severe anemia, and after five months' therapy his red cell count was up to 4,000,000.

In 1954 the patient was again hospitalized with severe anemia, submaxillary cellulitis of the left side of the face, and congestive heart failure. On this admission his stool was strongly positive for blood. His hemoglobin was 6.5 Gm., hematocrit 26 per cent, and white blood count 3,500 (differential normal). All bleeding and clotting tests were normal. After several transfusions and penicillin therapy, the lower left first premolar, which caused the submaxillary celluitis, was extracted under local anesthesia. Healing of the socket was uneventful, and no excessive bleeding followed the extraction.

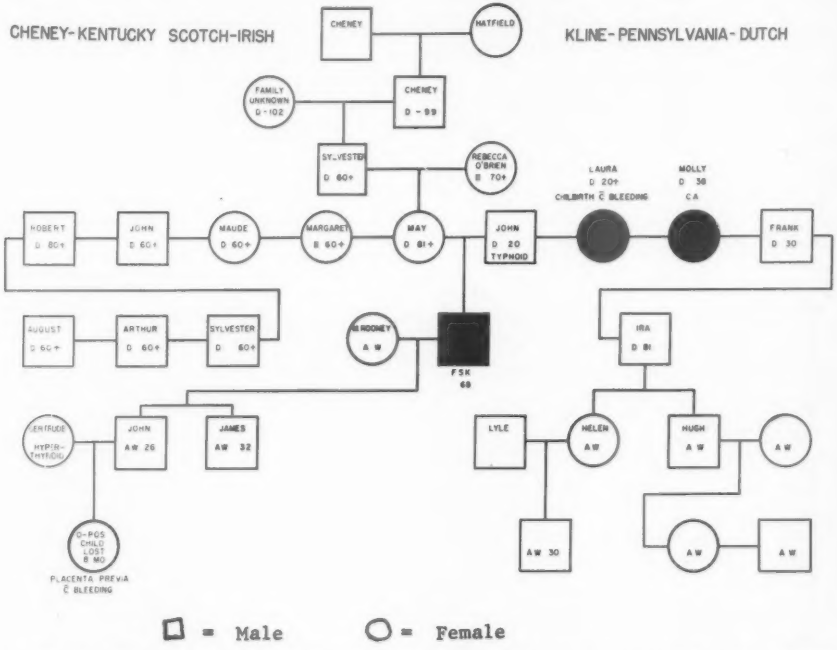

Fig. 4.—Family pedigree of Patient F. S. K. Blocked-out areas represent those in family with hereditary hemorrhagic telangiectasia. *D*, Deceased. *A.W.*, Alive and well. Numbers designate age of patient.

Various tests (gastroscopy, sigmoidoscopy, and barium enema) were performed to find the bleeding area, but no bleeding point was detected. It was felt that the patient's bleeding was somewhere in the small intestine. Upon discharge, the hemoglobin had been raised to 12 Gm. and the hematocrit to 44 per cent.

The patient was fairly well until March, 1956, when he suffered another heart attack and severe anemia. He was again hospitalized and received several packed-cell transfusions which, upon discharge, brought the hemoglobin to 13.5 Gm. His cardiac status responded to medical therapy.

Present Admission.—On the present admission his hemoglobin was 7 Gm., hematocrit 26 per cent, and white blood count 3,000 (differential normal). Blood chemistry on April

22, 1957, was as follows: total cholesterol, 109 mg./100 c.c.; alkaline phosphatase, 8.5 King-Armstrong units; total proteins, 5.7 mg./100 c.c.; albumin, 4.5 mg./100 c.c.; globulin, 1.2 mg./100 c.c.; calcium, 9.4 mg./100 c.c. Urine was normal, but stools were strongly positive for blood. All bleeding and clotting tests were normal.

Clinical examination of the oral cavity showed the oral mucous membranes and lips to be very pale. There were many punctate hemorrhagic areas on the buccal mucosa, hard and soft palate, tongue, and lips. The skin lesions were also punctate in appearance and were seen on tips of the fingers, face, ears, and neck, and a few small punctate hemorrhagic areas were found on the feet and trunk.

Microscopic examination of blood at various times showed a consistent poikilocytosis of red cells and a marked hypochromia. Radioisotope study was done with red blood cells labeled with Cr_{51}. Examination showed a blood loss of approximately 50 c.c. of blood during forty-eight hours. Study showed that there was no evidence of an active hemolytic process. Liver and biliary tests were taken, and all were approximately normal. Sigmoidoscopy and gastroscopy did not reveal bleeding points or a telangiectasia, and again it was felt that bleeding was in the small intestine. The patient received twenty-two packed-cell transfusions and upon discharge had an almost normal hemoglobin of 13.5 Gm./100 c.c. and a hematocrit of 44 per cent. Packed-cell transfusions were used because of his cardiac status.

During the present admission the patient had several teeth filled with no anesthesia, and a scaling and curettage was completed with no difficulty and with normal tissue response. Radiographs showed no abnormal bone condition.

Family History.—The patient's father died of typhoid fever at an early age. His mother died of undetermined cause at about 80 years of age. The patient was an only child. He had two children, both living and well. His aunt (father's sister) used to have frequent nosebleeds but died at an early age (34) of cancer. The family's genealogical chart is shown in Fig. 4.

CONCLUSIONS

The lesion or telangiectasia may cause little or no inconvenience (except a cosmetic one) when no bleeding occurs, but at about the fourth to the fifth decade the result of frequent bleeding and hemorrhagic episodes produces anemia of various degrees of severity. It is the anemia that brings about the debility in the patient.

Epistaxis can best be treated by topical hemostatic agents that can be applied to a bleeding point without having to be removed after bleeding has stopped. Oxycel (oxidized cellulose) and Gelfoam (absorbable gelatin sponge) fall into the latter category and are the ideal agents for use in the control of bleeding. Electrocoagulation also seems to be an effective method. Escharotic agents (silver nitrate, chromic acid) are used but are most effective on only a small bleeding point. According to Wintrobe,[5] electrocoagulation has proved satisfactory in stopping bleeding about the lips, the oral cavity, and the cutaneous surface of the body.

SUMMARY

A case of hereditary hemorrhagic telangiectasia in a 69-year-old man is reported. This is a first report of a unique case of hereditary hemorrhagic telangiectasia in the dental literature. It should be noted that the patient was able to receive dental treatment with no untoward reactions.

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PHARMACOLOGY AND THERAPEUTICS

THE USE OF CLORPACTIN AS AN ADJUNCT TO ORAL HYGIENE IN A GROUP OF HANDICAPPED CHILDREN

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THE problem of maintaining adequate oral hygiene in handicapped children is often the most serious that the dentist has to contend with in treating these children. Special hygienic techniques have been tried successfully in some cases, but in a large number of severely handicapped children poor oral hygiene remains the most important factor in the high incidence of dental caries and gingival disease. Over the years many mouthwashes, ranging from tap water and hydrogen peroxide to antibiotic and oxygenated types, have been tried with insignificant results. Past experiences have shown that more stomatologic allergic symptoms are observed with the use of antibiotic rinses, lozenges, and troches than with the systemic use of antibiotics.

Of the recent agents that have been introduced to help improve oral hygiene, a mouthwash which possesses active chlorine in the form of hypochlorous acid (Clorpactin† WCS-60 in solution) has been reported to be of value where other oral hygiene measures were not feasible or were ineffective. Clorpactin WCS-60 (brand of monoxychlorosene) is a water-soluble white powder which forms dilute solutions at pH levels varying from 6.8 to 7.2. In solution, Clorpactin WCS-60 has a highly pronounced wetting penetration, as well as germicidal and fungicidal properties. Previous investigations indicate that it is nontoxic and nonirritating to the oral tissues.

This study was designed to evaluate the efficacy of a Clorpactin mouthwash as an adjunct to oral hygiene in a group of handicapped children. Arnim[2] in 1954 and more recently Hawes have suggested a unique method for evaluating the status of oral hygiene, in teaching oral hygiene through visual education. They used basic fuchsin, a bacterial and protein stain, in a 3.0 per cent tincture applied with a cotton applicator to the dentogingival areas of the teeth. The patient is asked to close his mouth for a few seconds. Then the mouth is rinsed thoroughly with water. The bacterial mucinous plaque remains temporarily stained with a light blue color, and the patient is shown the extent of the stained bacterial debris plaque in a hand mirror.

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†Clorpactin WCS-60 used in this study was supplied by the Kasdenol Corporation, Huntington Station, New York.

A modification of this technique was used in this study. It entailed the use of a 1.0 per cent solution of basic fuchsin in aqueous alcohol (10 per cent) to avoid staining of synthetic restorations. The resultant stain fades in the mouth within an hour or so, or it can be brushed away. Kodachrome photographs were taken immediately after the above procedure was followed for each patient to ascertain the extent of the existing bacterial plaques before the study began. No attempts were made to alter the oral hygiene measures during the investigation. The same procedure and new Kodachrome photographs were repeated sixty and ninety days after the patients began using Clorpactin mouthwash. The parents of the patients were instructed to place one-half measure of white powder supplied into an 8 ounce glass of lukewarm water. Patients were to rinse their mouths for five minutes twice daily (after breakfast and after dinner).

As a pilot study, sixty-two handicapped patients, aged 6 to 19 years, were selected from the dental clinics of the Department of Dental and Oral Surgery of The Long Island College Hospital and The Kings County Medical Center to determine the value of this drug. These patients showed varying degrees of dental caries and gingival disease with consistent poor oral hygiene (as indicated in a previous study by the author). Plaques, calculus, and materia alba, as well as fetid odor, were present in the majority of the patients. A limited number (ten) of these patients were using tap water and hydrogen peroxide in equal parts for rinsing twice daily (after breakfast and after dinner). These patients served as controls, initially, and then as experimental subjects (for sixty days) using the same procedure that the entire group used.

	AVERAGE PER CENT OF PLAQUE REMOVAL	
	60 DAYS	90 DAYS
Hydrogen peroxide (3%) diluted with equal parts of water	30	No significant change
0.25% Clorpactin WCS-60 solution	60	90

Chart 1.

RESULTS

The patients who used Clorpactin mouthwash showed a marked improvement in oral hygiene, as studied by the above technique. The original plaque was reduced, on the average, 90 per cent by the termination of the study. As incidental results, there was a great reduction of fetid odor and gingival hemorrhage in many cases (confirming some similar observations made by another investigator on the use of Clorpactin in gingival disease).

The patients in the control group, using tap water and hydrogen peroxide as a rinse for the first sixty days, exhibited some improvement (evidenced by reductions up to one-third in several cases of the stained plaques). The difference between the experimental and the control groups was marked in all cases. The efficiency of the Clorpactin mouthwash as an adjunct to oral hygiene was greatest in the cases of very poor oral hygiene.

Fig 1.

Fig. 2.

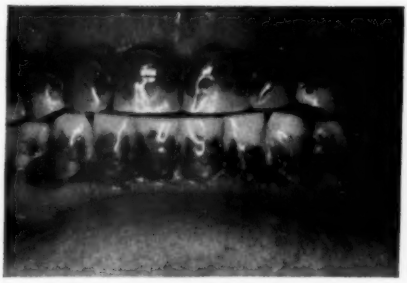

Fig. 3.

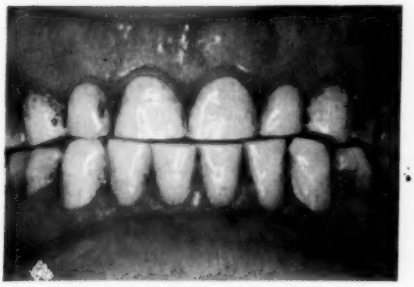

Fig. 4.

Fig. 1.—Original status of oral hygiene. Stained with 1.0 per cent tincture of basic fuchsin.

Fig. 2.—After sixty days of hydrogen peroxide 3 per cent solution. Stained with 1.0 per cent tincture of basic fuchsin.

Fig. 3.—After sixty days of Clorpactin WCS-60 solution. Stained with 1.0 per cent tincture of basic fuchsin.

Fig. 4.—After ninety days of Clorpactin WCS-60 solution. Stained with 1.0 per cent tincture of basic fuchsin.

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ORAL PATHOLOGY

A CASE OF AMELOBLASTOMA EVIDENTLY UNDERGOING TRANSFORMATION TO A NEW TYPE OF TUMOR

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IN THE limited literature on ameloblastoma at our disposal, I have not encountered any case similar to the one to be reported here. There are four main features of the case:

1. The gradual degeneration of the peripheral cuboid epithelium of the ameloblastic follicles and strands. Simultaneous with this change, most of the cells in the stellate reticulum showed proliferative activity and began to dissociate from each other and wander singly in the connective tissue stroma.

2. In some areas of the tumor mass, the tumor cells appeared to be densely distributed again with a tendency to assume squamous arrangement. A good number of the tumor cells now contained what we believe to be melanin granules.

3. In certain areas, the tumor cells which were densely distributed were now attacked by numerous and unusually large osteoclasts. The tumor cells engulfed by the osteoclasts appeared to be vital cells.

4. Some tumor cells appeared to be inside some of the blood vessels, indicative of metastases. In some blood vessels, cells like plasma cells with one or two nuclei were present. They seemed to be derived from the endothelial cells which were detached from the vessel wall.

CASE REPORT

The specimen used in this report came from a male patient, 17 years of age. It is regretted that we were unable to secure a satisfactory history of the case. According to the uncle who accompanied him, the patient fell from a hammock when he was about 1 year old. Swelling in the region of the chin was then observed. A few years later the lower anterior teeth erupted, but they were soft and were easily exfoliated. Whether or not the swelling

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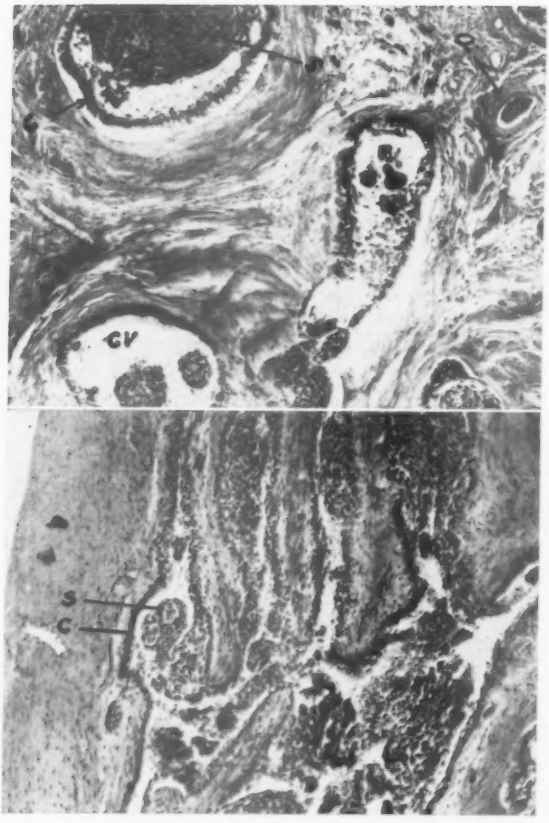
was inflammatory could not be ascertained. Fig. 1 shows a radiograph of the case before surgical operation. It may be noted that the new growth is located in the anterior region of the mandible. Because of the long duration of the lesion and because of radiographic findings, the case was tentatively diagnosed as ameloblastoma in the anterior portion of the mandible. The growth was surgically removed, and decalcified sections of certain portions of the specimen were made for microscopic examination.

Microscopic Findings.—Fig. 2, *A* shows ameloblastic follicles evidently undergoing cystic formation due to degeneration of the stellate reticulum. The peripheral cuboid epithelium, however, is still present. Fig. 2, *B* shows strands of ameloblastic cells. Although there are signs of partial degeneration of the stellate reticulum, the peripheral cuboid epithelium is still markedly present. These two findings strongly suggest that the case under consideration is one of ameloblastoma. Fig. 2, *C* shows the gradual disappearance of the peripheral cuboid epithelium and the tendency of the stellate reticulum to proliferate actively, as evidenced by the highly chromatic appearance. Fig. 2, *D* shows a higher magnification of a certain area of the stellate reticulum. It may be noted that some cells are undergoing mitosis. Fig. 3, *A* shows another section in which the stellate reticulum is completely devoid of the peripheral cuboid epithelium. It may be noticed that the cells are deeply stained and have the tendency to dissociate from each other and to wander singly in the stroma. Fig. 3, *B* is another illustration of deeply stained cells wandering singly in the stroma. In some areas of the tumor mass, these tumor cells are densely distributed again (Fig. 4, *A*). Fig. 4, *B* is a higher magnification of a certain area of Fig. 4, *A* showing the blood vessel surrounded by the tumor cells with a tendency to assume squamous arrangement. Some of the tumor cells appear to be inside the blood vessel.

Fig. 1.—Radiograph of the new growth before surgical operation. Note that the lesion is located in the anterior portion of the mandible. White spots are artifacts.

Another interesting feature of the case is the presence of numerous osteoclasts, evidently engulfing the tumor cells in a certain area of the tumor mass (Fig. 5, *A*). Some of the tumor cells contain what we believe to be melanin granules. Fig. 5, *B* shows, in higher magnification, osteoclasts engulfing the tumor cells. Some of the tumor cells appear to be inside a dilated capillary and attacked by the osteoclasts (Fig. 5, *C*). From their appearance, it seems that the tumor cells are not dead cells.

A.



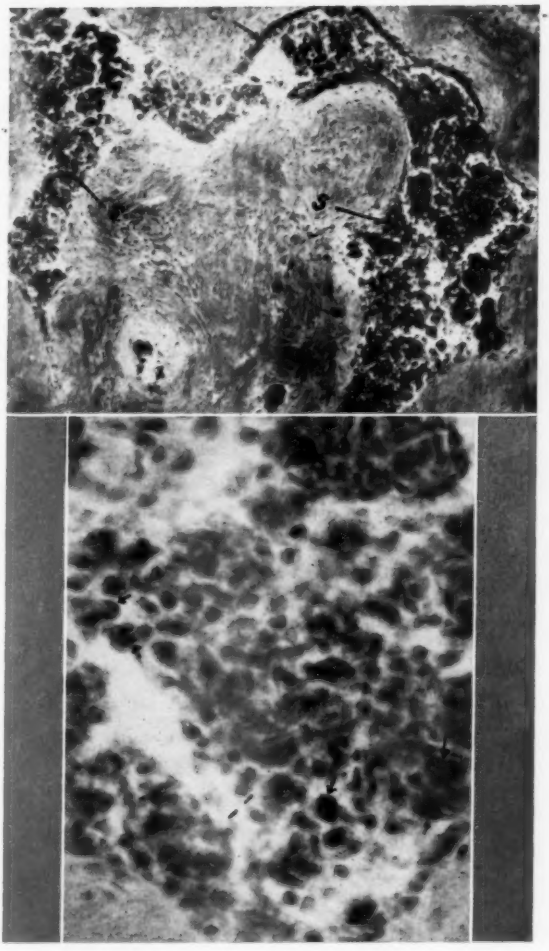
B.

Fig. 2.—*A*, Ameloblastic follicles undergoing cystic formation. *c*, Peripheral cuboid epithelium; *s*, stellate reticulum; *cv*, cystic cavity. Note tumor cells marked *a* apparently inside a blood vessel.

B, Ameloblastic strands showing partial degeneration of the cells in the stellate reticulum. *c*, Peripheral cuboid epithelium; *s*, stellate reticulum.

(*Continued on opposite page.*)

C.

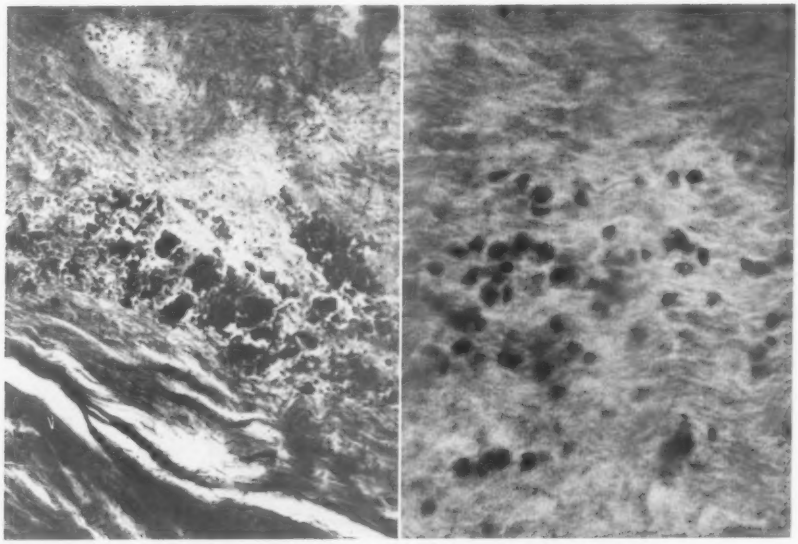


D.

Fig. 2. (Cont'd).—*C*, Peripheral cuboid epithelium (*c*) undergoing degeneration. Note the cells in the stellate reticulum (*s*) which stain deeply with hematoxylin and eosin.

D, A higher magnification of certain area of the stellate reticulum in C. Note mitotic figures indicated by arrows.

It has been hinted previously that some tumor cells appear to be inside a blood vessel. Fig. 6, *A* shows, in low magnification, a blood vessel evidently containing tumor cells. We have noticed in this particular case of ameloblastoma the presence of ovoid cells with eccentric nuclei similar to plasma cells. They seem to be endothelial cells detached from the vessel wall. They can easily be distinguished from the tumor cells because their cytoplasm takes a deep pink stain with hematoxylin and eosin staining and red with Mallory's triple stain. The tumor cells take a light violet stain in hematoxylin and eosin staining.



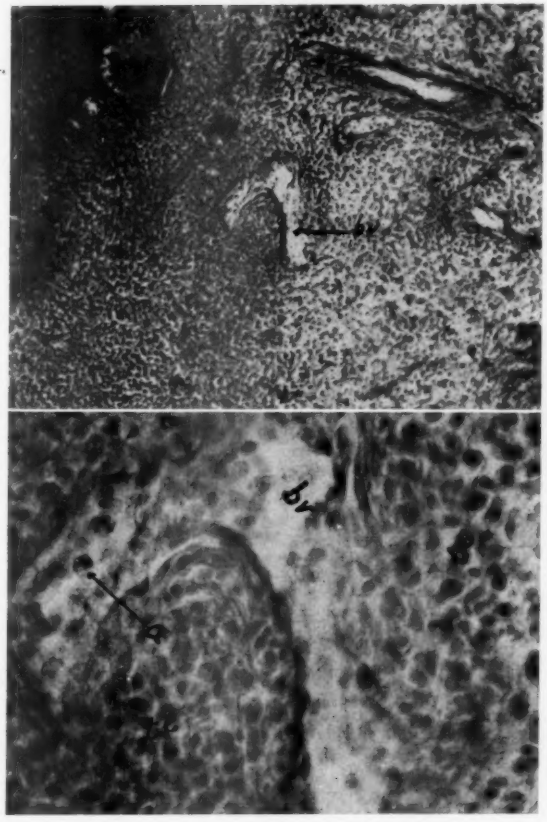
A. *B.*

Fig. 3.—*A*, Cells of the stellate reticulum completely devoid of the peripheral cuboid epithelium. Note that the cells are highy chromatic and show a tendency to dissociate from each other and wander singly in the connective tissue stroma.

B, Another area showing, in higher magnification, individual tumor cells scattered in the stroma. Note their highly chromatic appearance.

Fig. 6, *B* is another section showing, in higher magnification, tumor cells evidently inside the blood vessel. They can be easily distinguished from the plasmalike cell because of their morphology and staining reaction. Fig. 6, *C* is another area showing a blood vessel surrounded by tumor cells. It may be noted that the vessel is cut transversely in the upper end and diagonally in the lower end. Tumor cells are evidently present in the blood vessel. Fig. 7 shows a higher magnification of the blood vessel evidently containing tumor cells. There seems to be no doubt that this is a blood vessel, as evidenced by the presence of some red blood cells. We have noticed in different sections structures resembling a cross section of a blood vessel containing tumor cells like the structure marked a in Fig. 2, *A*. There is a possibility however, that the vessellike structure may be only the hyalinized part of the connective tissue which is often observed as a halo surrounding an ameloblastic follicle. However, we are presenting here (Fig. 8) a structure resembling a cross section of a blood vessel containing a mass of tumor cells. The endothelial cells, marked *e*, lining the vessel appear to be present. One endothelial cell detached from the vessel wall, marked *d*, appears now like a plasma cell.

A.

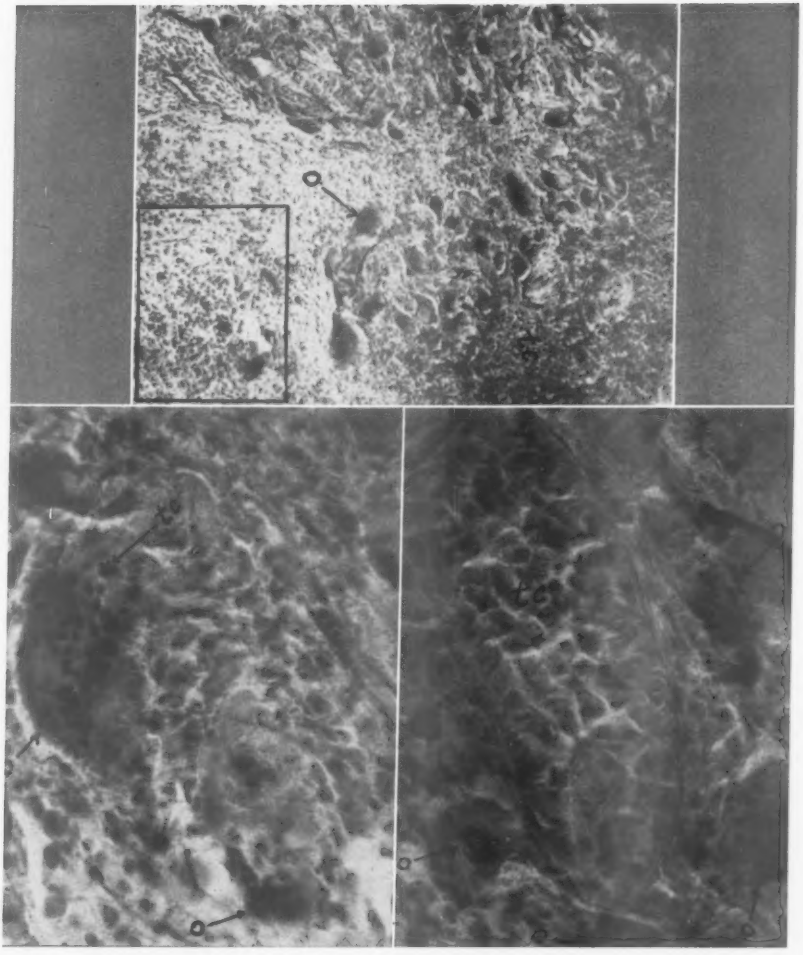


B.

Fig. 4.—*A*, Certain area of the tumor mass where the tumor cells are again densely distributed. Some of the cells contain melanin granules. *bv*, Blood vessel.

B, A higher magnification of the area close to the blood vessel in *B*. Note the tendency of the tumor cells (*tc*) to assume squamous arrangement. Some of the cells appear to be inside the blood vessel (*bv*). *e*, Endothelial cells; *a*, plasmalike cell evidently derived from endothelial cell.

A.

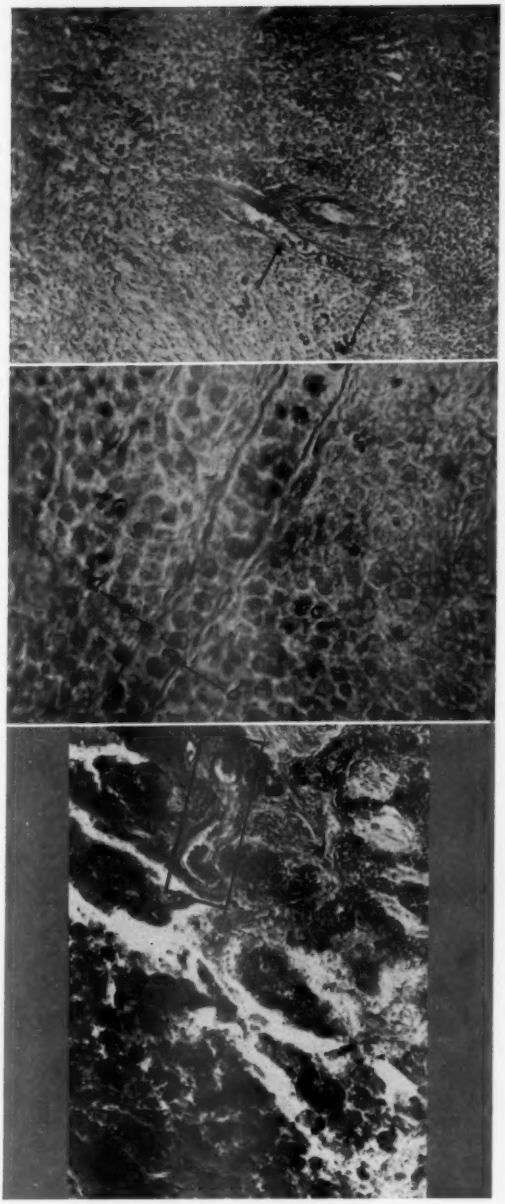


B. *C.*

Fig. 5.—*A*, A certain area of the tumor mass showing numerous osteoclasts, (*o*) attacking the tumor cells (*tc*). Tumor cells enclosed by a rectangle contain melanin granules.

B, A higher magnification of the osteoclasts (*o*) enguifing the tumor cells. One tumor cell (*tc*) appears to be vital with intact nucleus.

C, A higher magnification of tumor cells (*tc*) evidently inside a dilated portion of a capillary and attacked by osteoclasts (*o*).



A.

B.

C.

Fig. 6.—*A*, A longitudinal section of a blood vessel (*bv*) showing, in low magnification, tumor cells (*tc*) evidently inside the vessel. Many tumor cells in area (*A*) contain melanin granules.

B, Another blood vessel showing, in higher magnification, tumor cells evidently inside the vessel. *a*, Tumor cells inside the blood vessel; *b*, plasmalike cells possibly derived from the endothelial cells; *tc*, tumor cells outside the blood vessel.

C, Another area of the tumor mass showing a blood vessel (enclosed by a rectangle) of which the upper end appears to be cut transversely and the lower portion somewhat diagonally. In both ends, tumor cells appear to be inside the vessel.

Fig. 7.

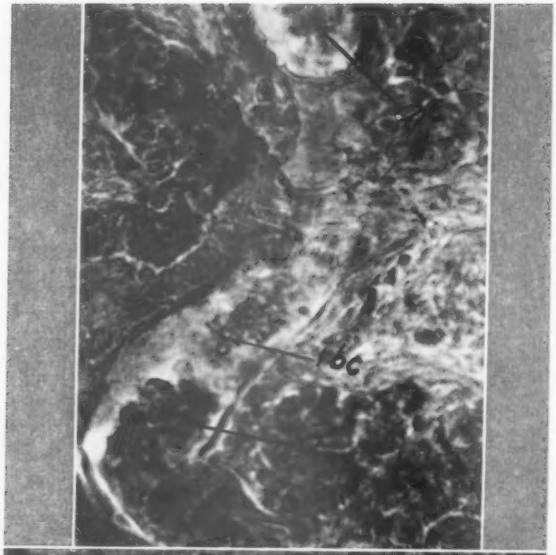


Fig. 8.

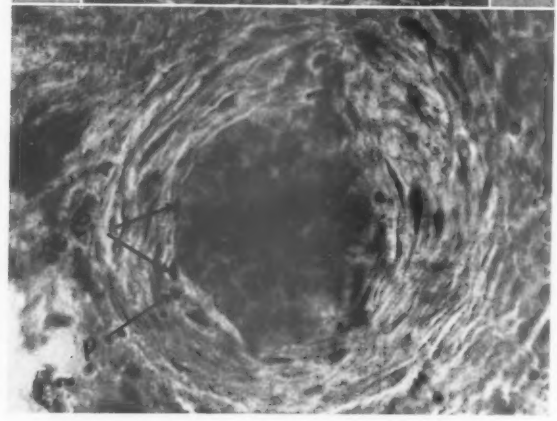


Fig. 7.—A higher magnification of the blood vessel shown in Fig. 6, *C*. *tc*, Tumor cells outside the blood vessel; *a*, masses of tumor cells evidently inside the blood vessel; *rbc*, red blood cells.

Fig. 8.—A higher magnification evidently of a transverse section of a blood vessel, possibly a vein containing at the center a mass of tumor cells. *e*, Endothelial cells; *p*, plasmalike cell.

COMMENT

The question as to whether or not an ameloblastoma may become malignant may be considered still controversial, in spite of some reports to the contrary. The present case reported, however, may shed further light on the question. The proliferation of the cells from the stellate reticulum, which later dissociate from each other to be distributed over the stroma, seems to give a picture similar to the "acute" or "inflammatory" carcinoma of the breast as described by Foot.[1] If our finding of tumor cells inside some blood vessels gains general acceptance, this may be considered as another point in favor of a view that the apparent change is toward malignancy. It is quite difficult to explain the presence of osteoclasts engulfing the tumor cells. As stated previously, the tumor cells appear to be vital. In tumors of the jaws, the presence of osteoclasts is frequently observed, but not to engulf or attack the tumor cells. These osteoclasts are evidently present to resorb the bone.

The author acknowledges the courtesy extended by Jaime Laico, M.D., F.P.C.S., Assistant Professor of Surgery, College of Medicine and Professorial Lecturer of Oral Surgery, College of Dentistry, University of the Philippines, who supplied the specimen used in this report.

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PALATAL ANEURYSM

Review of the Literature and Report of a Case

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INTRODUCTION

ARTERIOVENOUS aneurysms involving the palate are extremely rare. Some cases have been reported in the dental literature, however. The purpose of this article is to review the literature and to describe an unusual aneurysm involving the palate.

REVIEW OF THE LITERATURE

In 1841 Hullihen[1] treated a pregnant female patient for what he called an "aneurism by anastomosis of superior maxillare." After reviewing his article, we feel that he was confronted with a pregnancy tumor and not an aneurysm. The tumor was comprised of a mass on the labial and lingual surfaces of the maxillary central incisors and was joined by an isthmus of tissue. Hullihen reported that it was "of a dark purple color, very soft, and pulsated as distinctly as the artery at the wrist." The mass was ligated, and "on the sixth day the tumor came away, and not a vestige of the disease remained."

Ten years later, in January, 1851, Arms[2] was consulted by a 35-year-old woman concerning a tumor located behind the upper central incisors. "Artificial crowns had been engrafted two or three years previous" on three of the upper incisors. The tumor was reported to be about the size of a large almond, red to purple in color, vascular, and moderately soft. The patient had spasmodically painful, and occasionally hemorrhagic, episodes with the mass. The area was incised and a copious flow of blood followed. The upper teeth were removed in an attempt to get to the base of the mass. Upon examination after the teeth were removed, the base of the tumor was visualized and ligated with immediate cessation of hemorrhage. "The patient was requested not to disturb the ligature, but suffer it to come away of itself, which it did in some three or four days without any subsequent hemorrhage."

Teirlinck[3] reported an "aneurism of the superior palatine artery" in a 74-year-old man. "The tumor occupied the roof of the palate, which bled so frequently that the patient was much exhausted. The tumor was soft, elastic

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and pulsated synchronously with the heart, alternately expanding and diminishing. Its cause was unknown, and it had lasted for three weeks. The actual cautery was employed, the slough separated in eight days, the hemorrhage did not recur, and a perfect cure resulted."

In 1883, Dubrueil[4] reported a case in which he had examined a 38-year-old woman who complained of a swelling in the left soft palate which had increased in size for some six months. It extended about 2 cm. in an anteroposterior direction and involved the whole left half of the soft palate. The tumor was of normal color and was neither fluctuant nor pulsating. At the time of surgery, massive hemorrhage necessitated ligation of the common carotid artery. When the patient recovered consciousness, she was aphonic. Secondary hemorrhage occurred two and one-half hours after the original surgery, necessitating religation of the common carotid and ligation of the superior thyroid and both the internal and external carotid arteries. When the patient recovered consciousness, she was not only aphonic but very deaf and the left pupil was greatly contracted. Some days later the patient was seized with left-sided hemiplegia and died soon thereafter. At the postmortem examination, it was found that the tumor communicated directly with the internal carotid artery. "The tumour was evidently an aneurism of the internal carotid at a point on a level with the palate, which had destroyed the wall of the pharynx and gradually filled the velum."

A 26-year-old man was referred to Marshall[5] on Dec. 26, 1889, for treatment of a 1 by 1½ inch egg-shaped swelling involving the right alveolus. All maxillary teeth, except the two central incisors, had been extracted some eighteen to twenty months before this examination; the extractions had been accompanied by a great deal of trauma. The mass, with the small end anteriorly, extended from the alveolar process to the median line and from the maxillary tuberosity region to the cuspid region. "In character it was soft, fluctuating, compressible, and with very marked pulsation. In color, it was slightly deeper in tint than the surrounding mucous membrane. Upon puncturing it with an exploring needle, a jet of arterial blood followed its withdrawal, and continued to spurt for about half a minute, when the hemorrhage ceased. The diagnosis was aneurismal tumor of the posterior palatine artery, with possible anastomosis with some branch of the superior maxillary artery, the result of injury in the extraction of the teeth." It was decided to sclerose the tumor, and this was done. When the slough had occurred, it was found that the aneurysm "occupied the antrum of Highmore, and had produced absorption of the palatine process of the superior maxillary bone and the nasal wall of the antrum, leaving a large opening into the nasal fossa." Eventually the opening into the antrum and the nasal fossa closed without any further difficulty.

In 1895, Edward[6] reported an aneurysm involving the anterior palatine artery. The patient was an elderly man for whom a "partial set of artificial teeth, to complete the superior maxillary row" had been constructed. Several weeks after the denture was inserted, a small vesicle appeared "upon the lingual centre of the roof of the mouth." The mass was the size of a large pea, purplish in color, and exhibited no pulsation. The mass was excised with scissors, following which there was a copious flow of blood from the incisal

foramen. To control the hemorrhage, a piece of cork made into the shape of an "X" was forced into the "palatine hole." "On the fourth day after its insertion, the plug came away, and the patient experienced no further difficulty or inconvenience." The base of the denture may have irritated the anterior palatine artery, thus initiating the formation of an aneurysmal sac.

Shield,[7] in 1896, reported a case of a 57-year-old man who had an aneurysm of the left hard palate. The etiology and time of origin of this deformity were unknown to the patient. The mass had an ulcerous type of lesion that eroded into the vascular supply beneath and permitted blood to be "issued forth in jets." Surgical therapy was elected, and the patient underwent a hemisection of the left maxilla. It is interesting to note that the microscopic examination of the specimen was discussed but the patient's postoperative course was not mentioned.

Also in 1896, Freeman[8] described the case of a 35-year-old man who had a mass the size of a chestnut situated in the palate. The mass was fluctuant but had no pulsations and was thought to be an abscess from the maxillary lateral tooth. A copious flow of blood followed an incision of the mass. "The treatment in this instance, after using a solution of persulphate of iron, which did not prove a permanent coagulant, was to wipe the entire inner surface of the sac with equal parts of the tincture of iodine and a 95 per cent solution of carbolic acid. This proved a good, sufficient coagulant, which arrested the hemorrhage permanently." Freeman suggested the use of an aspirating needle for a differential diagnosis. There was no recurrence of the aneurysm after eighteen months.

In 1896, Goslee[9] presented a patient with a large aneurysm involving the posterior palatine artery. He demonstrated a hinged-denture type of prosthesis which he had constructed to reduce the long-standing aneurysm.

The last article appearing in the dental literature on palatal aneurysms was written by Peach[10] and published in a 1905-1906 journal. Peach removed a maxillary left second molar, and within twenty-four hours a throbbing mass the size of a marble developed at the extraction site. Thinking the mass to be pus, he incised it and liberated a stream of arterial blood. Immediate consultation was obtained and they "dissected down the overlying tissue, placed the artery back in its channel, supported it there, and until I lost sight of it some years after, there was no recurrence of trouble."

CASE REPORT

The first examination of this patient was at Lawson VA Hospital in Atlanta, Georgia. The patient, a 31-year-old white man, was admitted to the hospital in 1948 because of a swelling in the palate. For several days prior to admission, this swelling had caused pain in the left side of the face.

The patient stated that when he was 2 years of age he had an overhanging lesion involving the left maxilla, extending down over the teeth. The lesion was anterior and buccal to the site of the present swelling. It hemorrhaged easily, and the bleeding was difficult to arrest. This mass was treated with three radium or x-ray treatments. The last treatment was given in 1922, and the lesion disappeared with no further complications. The present palatal lesion was noted first while the patient was receiving a routine oral examination in the Army.

There were five periods of hospitalization concerned with the management of this palatal swelling. The first examination revealed a tumorlike mass in the palate, extending from the anterior gingiva to the posterior palatine area. It involved the hard and soft palates to the midline, measuring 3.3 by 5 cm. It was bluish tinged and there was a vigorous pulsation on palpation. The mucosa overlying the area was thinned. X-ray studies revealed a circumscribed area of soft-tissue density resembling a cyst or mucocele superimposed on the left side of the palate, as well as a cloudy left maxillary antrum.

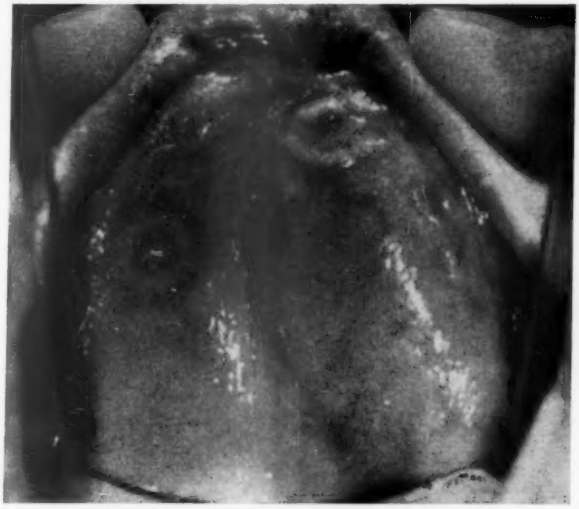

Fig. 1.—A soft, smooth, pulsating aneurysm, measuring 1½ by 3 inches, covering the left hard palate. It extends from the anterior alveolar crest to the posterior border of the hard palate and from the midline to the crest of the alveolar ridge.

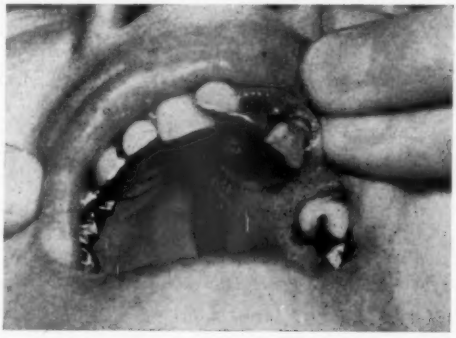

Fig. 2.—The palatal aneurysm as it appeared in March, 1948. The dark shadows seen in the oral cavity were produced by the photographic lighting.

No evidence of erosion or any destructive change in any of the bones of the skull was shown at that time. The mass was aspirated with a 20 gauge needle, but apparently the aneurysm was not entered since only a small amount of submucosal hemorrhage was obtained. Dental treatment at this time consisted of restorations and periodontal therapy.

The patient was hospitalized again several weeks after the first admission. During this admission he was presented to the Tumor Board, and it was the opinion of the Board that this was an arteriovenous aneurysm. It was reccommended that no extractions be done unless essential.

The patient was admitted to the hospital again in April, 1956, complaining of pain in the area of the left palate. Pain also involved a number of carious teeth. The patient was again presented to the Tumor Board, and the previous diagnosis of arteriovenous aneurysm was confirmed. It was recommended that the remaining teeth be extracted (Fig. 3). It was decided to reaspirate the mass to ascertain the amount of pressure exerted and the color of the aspirated blood. The mass was re-entered with a 20 gauge needle, and 2 c.c. of bright red blood were aspirated immediately. The patient refused to have the aneurysm removed at the time of the extractions. The remaining teeth were extracted and alveoloplasty was performed without any complications. The patient had no bleeding problems and made an uneventful recovery. He was discharged to return later for construction of full dentures.

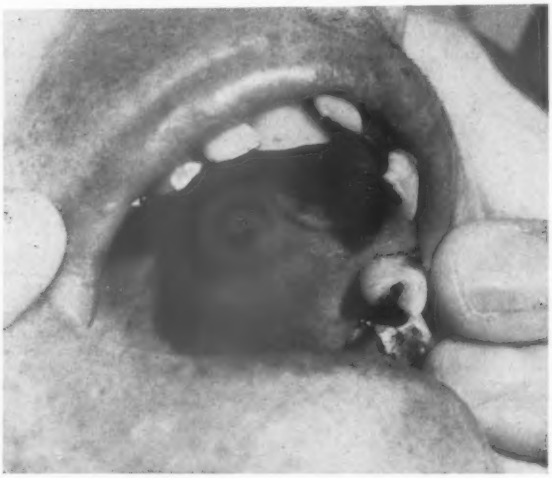

Fig. 3.—The aneurysm as it appeared in April, 1956, prior to extraction of teeth. The size of the swelling has not changed noticeably since 1948. The dark shadow seen in the midline is a photographic shadow.

The patient was readmitted to the hospital in August, 1956, for construction of dentures. At this time it was noted that there seemed to be some extension of the mass over the edentulous ridge on the left. The patient was again advised to consent to the removal of the aneurysm, but again he refused. Dentures were inserted with indicated relief over the mass. The patient was discharged and requested to return in thirty days for a check on toleration of the prosthesis. He reported in October and was very well pleased with the result obtained. The patient was advised to have periodic examinations.

DISCUSSION

One can only speculate as to the nature of this patient's childhood lesion. The patient merely knows that the mass hemorrhaged easily and that it was difficult to arrest the flow of blood. The physician who treated the initial lesion died many years ago, and his records could not be located. The most

likely diagnosis of the initial lesion seems to be a hemangioma. The relationship, if any, which that lesion and its treatment could have on the present mass is undetermined.

The descending palatine artery divides into major and minor arteries in the pterygopalatine canal. The major palatine artery emerges from the pterygopalatine canal through the major palatine foramen. It then runs anteriorly along the medial side of the maxillary alveolus to the incisive canal. The palatal swelling in this patient was pulsating and followed the same anatomic pathway; therefore, it is felt that this mass was an aneurysm of the major palatine artery.

There are several methods by which this unusual problem could be managed. A conservative but less effective method would be to construct dentures only, with adequate relief over the enlarged area. Possibly, the aneurysm could be reduced in size by slowly adding to the denture base over the enlarged area; however, trauma of any sort could initiate massive arterial hemorrhage with the possibility of exsanguination. Finally, the mass might increase to a degree where it would become inoperable.

It would seem, however, that the most obvious and most desirable treatment would be surgical removal of the aneurysm. Had it been possible to attack the problem surgically, several possibilities might have been considered. Although not demonstrated radiographically, the prolonged, pulsating pressure may have enlarged the pterygopalatine canal, allowing the aneurysm to extend upward into the canal. If this had happened, ligation of the artery might have been more difficult. In all probability, however, the aneurysm could have been ligated and removed surgically with a minimum of difficulty. Consideration was given to other surgical methods, such as ligation of the external carotid artery. The probable existence of a collateral blood supply had to be considered. It is academic to do any more than speculate on the surgical procedure of choice; the procedure would depend on conditions found when the aneurysm was exposed.

It is interesting to note that this patient had undergone exodontia, without complications to date, from an area that reportedly was previously exposed to x-ray or radium therapy. There was no clinical or radiographic appearance of osteoradionecrosis.

SUMMARY

The literature on palatal aneurysms has been reviewed. A new case of palatal aneurysm has been described and added to the existing literature.

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BENIGN FIBROEPITHELIOMA OF THE HARD PALATE

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FIBROEPITHELIOMA is a benign tumor composed of many cystic structures lined with squamous epithelium and containing laminated keratin. An abundant amount of fibrous tissue stroma is present. The tumor is completely encapsulated, smooth, and firm in consistency.

The following case is reported because of the unusual cellular structures found in a fibroepithelioma.

CASE REPORT

A 62-year-old man came to the dental clinic of the St. Francis General Hospital complaining of severe pain in the roof of his mouth.

Intraoral examination revealed a tumor mass on the left side of the hard palate in the molar area. A small area of ulceration was noted on the surface, the periphery of which was slightly elevated and inflamed. The remaining mucous membrane covering the mass and the adjacent tissues appeared to be normal. The tumor was firm and nonmovable. Only four upper anterior teeth were present; they showed severe periodontal infection and extensive dental caries. The lower jaw was edentulous. All remaining oral structures appeared essentially negative.

Extraoral examination was negative except for the presence of slightly enlarged bilateral lymph nodes.

Roentgenographic Examination.—An ill-defined area of decreased density was revealed in the hard palate; the margins were not well defined. A tentative diagnosis of palatal abscess was made.

The patient was admitted to the hospital for excision of the mass and extraction of the remaining upper teeth. Physical examination revealed a well-nourished Negro man in no apparent physical distress other than the pain present in the oral cavity. Physical findings were essentially negative. All laboratory, blood, and urine tests were within normal limits.

Operation.—The patient was anesthetized with Pentothal sodium supplemented by endotracheal nitrous oxide and oxygen. The four remaining upper teeth were extracted, and an alveolectomy was performed. This was followed by enucleation of the tumor mass from the hard palate. The tumor was shelled out easily and was found to be well encapsulated. It appeared to be about the size of a hazelnut. The diagnosis was changed at this time to benign fibroma of the hard palate, and the specimen was sent to the laboratory for routine microscopic examination.

Pathologic Examination.—The specimen consisted of three pieces of tissue from the hard palate, the largest of which measured 1.7 by 1.4 by 1.2 cm. The tissue was covered on one surface by a small elliptical piece of bluish gray mucous membrane. The undersurface was rounded and covered by a thin mottled red and yellow capsule. On section, this mass was found to be firm, granular, and grayish white with numerous flecks of bright yellow studding the cut surface.

Fig. 1.—Low-power section of fibroepithelioma.

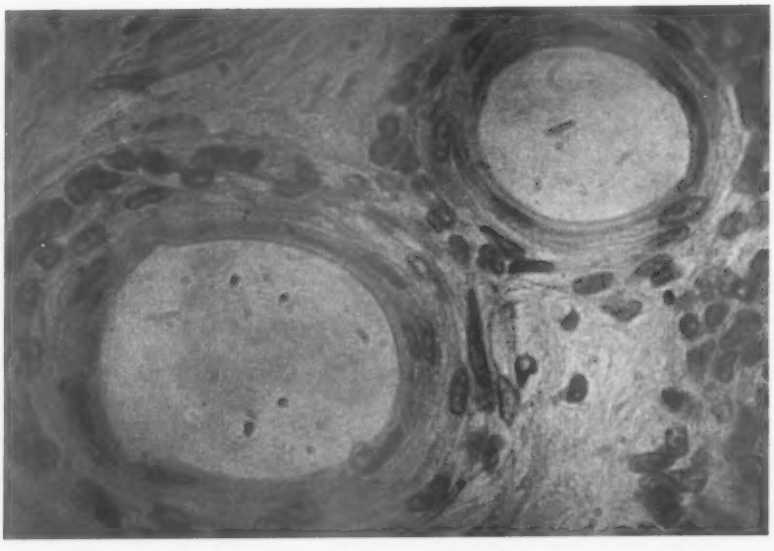

Fig. 2.—High-power photomicrograph showing cystic structures lined with squamous epithelium.

A section of tissue from the hard palate was covered on one surface by a thick layer of stratified squamous epithelium which was lightly keratinized (Fig. 1). Beneath the epithelial surface there was a well-circumscribed encapsulated mass covered by fibrous tissue. The mass itself was composed of many cystic structures lined by squamous epithelium (Fig. 2). The lumen of the cysts contained a large amount of coarsely laminated keratin. Between the cysts, which varied greatly in size, there was a large amount of dense fibrocollagenous tissue and a small amount of pale-staining adipose tissue. Throughout this dense stroma there were numerous focal collections of chronic inflammatory cells, chiefly lymphocytes, plasma cells, and an occasional eosinophil.

Pathologic diagnosis: Benign fibroepithelioma of the hard palate.*

Recovery was uneventful and the patient was discharged to go home. Postoperative examinations revealed satisfactory healing of the hard palate. Recurrence is not evident at this time; however, the patient will return for periodic examinations.

SUMMARY

A case of benign fibroepithelioma has been reported. A review of the available literature concerning fibromas and mixed tumors of the oral cavity has failed to reveal any tumor with the same constituent elements as those found in this particular case.

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*The pathologic reports were prepared by R. C. Hamilton, M.D., and Earl Davis, M.D., Department of Pathology, St. Francis General Hospital.

ENDODONTICS

MULTIPLE REPLANTATION OF ANTERIOR TEETH
Report of a Case

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At 1 P.M. on Dec. 9, 1956, a 15-year-old boy was struck by a toboggan and the maxillary left lateral and central incisors, right lateral incisor, and right cuspid were avulsed and lost in the snow (Figs. 1 and 2). The referring dentist, Dr. W. J. Smith, saw the patient within an hour and sent him to find the teeth in the snow. The boy was successful in his search and returned with the teeth. Each tooth was grasped by forceps which touched only the crown, while all particles of debris were removed from the root surface by using a camel's hair brush moistened in normal saline solution. The root was then covered with a layer of gauze saturated with normal saline solution. The root and gauze were

Fig. 1.

embedded in a block of plaster, leaving the crown and about 4 mm. of the apical third of the root disclosed (Fig. 3). About 2 mm. of the root was resected and the root canal was opened through the lingual surface of the crown (Fig. 4).

The root canal was enlarged to the diameter of a No. 6 file; all debris was removed, the canal disinfected by the use of phenolsulfonic acid and later neutralized with sodium hypochlorite (Fig. 5). A silver point was selected to fit the root canal and was locked into place in a cementing medium using the formula of L. I. Grossman (Fig. 6). The silver point was cut back to the en-

Fig. 2.

Fig. 3.

Fig. 4.

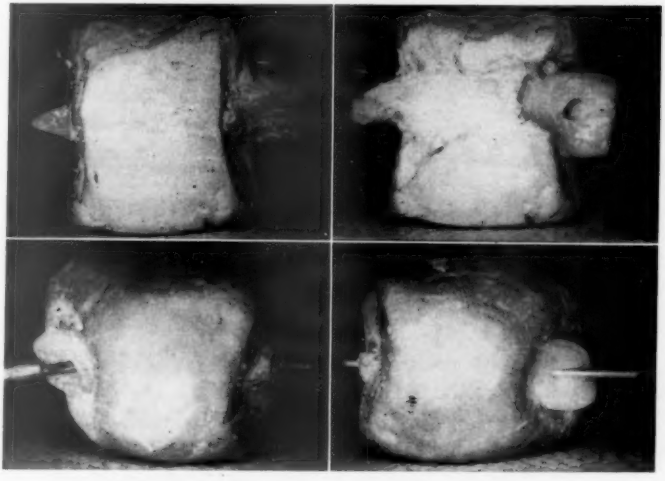

Fig. 5.

Fig. 6.

Fig. 7.

trance of the root canal, and the pulp chamber was filled with silicate cement to yield the maximum transmission of light (Fig. 7). An anesthetic was placed, and each socket was carefully cleansed of blood clot and debris. The teeth were gently returned to place, and silk sutures were used to aid in adapting the tissues into place (Fig. 8). A radiograph was taken to show the roots in position (Fig.

Fig. 8.

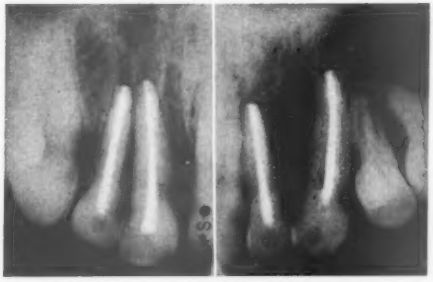

Fig. 9.

9). Dr. Smith now took an alginate impression of the maxillary teeth and poured a model in stone (Fig. 10). The services of an orthodontist were now conscripted, and Dr. M. R. Culbert completed a bite-plane type of splint at 11 P.M. on the same day (Figs. 11 and 12). When the patient was seen seven days after the accident (Fig. 13), the tissues were healing normally. The teeth were not unduly mobile in the sockets but continued to require support. Twenty-one days after the accident the splint was discarded. Thirty days after the accident the teeth were firm and symptomless. The tissues actually appeared to be much healthier than those in the anterior region of the mandible (Fig. 14). On Dec. 9, 1957, one year after the accident, the patient was again examined. The teeth were clinically symptomless; the tissues were excellent, with the exception of one small region of local irritation distal to the left central incisor caused by a deposit of calculus (Fig. 15). The radiograph showed good bone pattern in the periapical region and evidence of root resorption in the left central incisor alone. It will be noted that during the year the pulp of the right central incisor

Fig. 10.

Fig. 11.

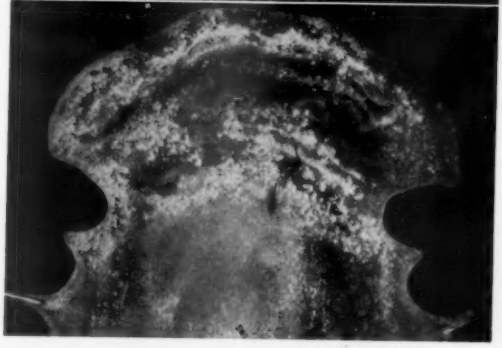

Fig. 12.

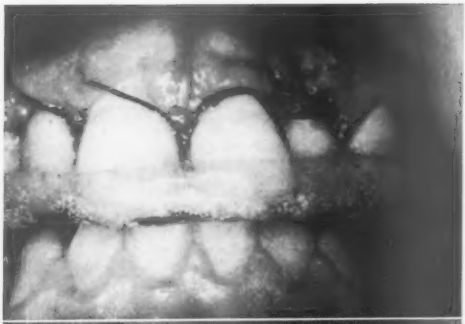

Fig. 13.

Fig. 14.

Fig. 15.

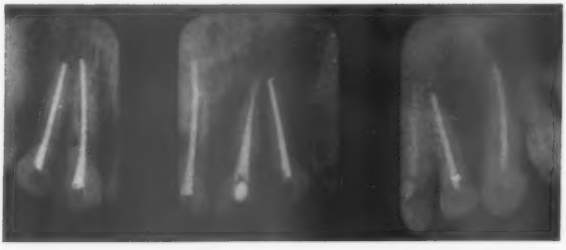

Fig. 16.

lost its vitality and the root canal was obturated (Fig. 16). Replantation of avulsed teeth is generally considered to be a temporary measure. This patient is now 17 years of age, and if these teeth can be preserved in a healthy state for even a few more years he will have passed through those formative years when the psychological impact of the loss of anterior teeth might mean the difference between success and failure in his social or business life. The life of the replanted tooth is often longer than we think.

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A COMPARATIVE STUDY OF THREE ROOT CANAL SEALING AGENTS

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(Continued from the September issue, page 1041)

CLINICAL STUDY

THE chief objective of applied research is to improve clinical application. With the accumulated evidence gathered through laboratory studies that the root canal sealing agents tested held promise, these agents were subjected to clinical use in human subjects. The Kerr sealer was not used clinically by me, since this agent has proved its value in countless thousands of cases treated over many years.

Method.—All teeth treated were first rendered free of infection and prepared for obturation. The technique of obturation varied, as indicated, in each case being treated. Wherever possible, the lateral condensation of gutta-percha was employed. In multirooted teeth, silver points were used. In the larger roots, silver points were occasionally used in conjunction with gutta-percha and lateral condensation techniques.

Where the lateral condensation technique was employed, the sealing agent was carried into the root canal on the first point that was inserted to the apex. Each of the additional points that were added to the root canal were also dipped into the root canal sealing agent.

When the single-point technique was employed, a power-driven instrument, the Lentula, was used to carry the sealing agent into the canal, and the canal itself was partially filled with the sealing agent. The selected point was then rolled in the sealing agent in question, carried to the apex, and wedged tightly into position.

In both instances, all excess root canal sealing material was removed from the pulp chamber of the tooth before placement of a final outer seal.

The actual root canal sealer was prepared on a sterile glass slab. When the new Grossman sealer was used, it was mixed to a creamy texture which would stretch approximately one inch from the slab before breaking. The Diaket was prepared in a similar manner. However, the manufacturer does provide a "spoon" and a special dropper bottle, so that a given quantity can be measured easily. When one spoonful of powder to one drop of liquid was measured

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twelve minutes of working time resulted. One spoonful of powder to two drops of liquid provided twenty minutes of working time. In these studies the twenty-minute working time was preferred.

Fig. 19.—*A*, Central incisor requiring root canal therapy.
B, Purposeful overfilling of canal with Diaket to study tissue tolerance.
C, One year later. Note healing of periapical tissues and lack of distortion of extruded Diaket sealer.

Results.—Teeth of thirty-seven patients obturated with the new Grossman sealer. They were evenly divided among the anterior and posterior teeth. Some of these teeth have been observed for almost one year. There have been

no unusual after effects that could be attributed to the sealing agent. Healing, as measured by clinical observation, and x-ray studies reveal good results. There is some evidence that excess root canal sealer is being slowly absorbed.

Three hundred seventy-two patients were treated, using the polyketone compound, Diaket, as the sealing agent. This was used, as stated, in conjunction with silver or gutta-percha points, as described. A total of 408 teeth was treated, with the following distribution:

Anterior teeth	152
Premolar teeth	100
Molar teeth	156

Some of these teeth have been under observation for a period of fourteen months. Among those patients who have returned for periodic examination, there is no evidence of resorption of excess root canal sealer, and healing is taking place normally.

Fig. 20.—Maxillary premolar tooth with canal filled with Diaket and lateral condensation of gutta-percha. Note flow of Diaket through lateral canal.

It was also noted that when Diaket had been forced beyond the apex of the tooth, either inadvertently or purposely, to test the ability of the tissues to tolerate it, where large areas existed, the bone seemed to envelop the extruded Diaket material.

DISCUSSION

Although the Kerr sealer has proved its value as a root canal sealing agent, it has several disadvantages:

1. It will discolor the tooth because of the silver and iodine salts that it contains.
2. The setting time is too rapid. Thus, in the cases of multi-rooted teeth, a fresh batch of sealer must be prepared for each canal.
3. It lacks the ability of retaining silver points in the canal during crown and bridge preparation.

4. As observed in the permeability studies, methylene blue will permeate the outer surface of this sealing agent.

The new Grossman sealer is superior to the Kerr Sealer in several respects:

1. It is a white substance and does not discolor the tooth.
2. It has better antimicrobial activity than the Kerr sealer.
3. It will hold the longer and wider points more securely than the Kerr sealer.

The Kerr sealer is more efficient than the Grossman sealer, however, in retaining the thinner, shorter points in the root canal. This is a serious shortcoming in the Grossman sealer. Another disadvantage of the Grossman sealer is a lack of uniformity in preparation. One pharmacy prepared a batch of the new Grossman sealer that would not set. After many trials, it was found that when the source of rosin was changed a better quality resulted. Another pharmacy had a far too rapid setting time in its preparation of the material. The working time was less than ten minutes in this instance.

The polyketone compound, Diaket, seems to be the most promising compound tested to date. It seems to come closest to the criteria for an ideal root canal sealing agent listed at the beginning of this article. Among its superior features are the following:

1. It is white and will not stain the tooth.
2. It has good antimicrobial activity.
3. It is well tolerated by the body tissues.
4. It can be used alone or in combination with silver or gutta-percha points.
5. It will hold these points more tenaciously than the other sealers tested.
6. With solvent, such as chloroform, it can be readily removed from the root canal if necessary.
7. It is completely impervious to methylene blue.
8. It has an excellent working time.
9. It will not dissolve in or be absorbed by the periapical fluids and tissues.

Earlier studies by Kluczka,[5] Schug-Kosters,[11] and Trauner[10] advocate the use of Diaket for obturation of the root canal following vital extirpation. They used the sealer alone in its paste form. Their studies did not include its use in infected pulpless teeth. The disadvantage of using the paste alone is the difficulty in completely filling the root canal with this method, together with the greater number of x-ray exposures that would be required in determining whether or not the canal were being filled.

SUMMARY

Three available root canal sealers were investigated and compared for tissue tolerance, antimicrobial activity, tensile strength, permeability, and clinical use.

The Kerr sealer, the new Grossman sealer, and Diaket were tolerated equally well by the soft tissues of the rabbit.

All three compounds possessed antimicrobial activity against the ten microorganisms studied. The new Grossman sealer and Diaket were somewhat more active in this respect than the Kerr sealer.

The Diaket was superior to the other sealers for its tensile strength and resistance to permeability.

The Kerr sealer has stood the test of time, as far as clinical use is concerned. The other two compounds are equally well accepted by the human periapical tissues, however, and have other more desirable properties.

The author wishes to express his appreciation to Dr. Herbert Cobe, Professor of Bacteriology, and Dr. Joseph McCunney, of the same department, for their assistance in the bacteriologic phase of this work.

The author also wishes to express his thanks to Dr. Frederick James, Professor of Pathology, for the preparation of the histologic sections; to the E. L. Conwell Co., Engineers, for their assistance in the tensile strength studies; and Mr. Alex Mucha, for his photography.

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RESEARCH

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DELAYED REIMPLANTATION OF SECOND MOLARS IN THE SYRIAN HAMSTER

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INTRODUCTION

DURING the past thirty years, a vast amount of experimental data have accumulated on tissue grafting of all kinds. Clinical application has been extended to almost every phase of medical practice. Patients survive critical burns today because of present knowledge of skin grafting; others see by means of corneal transplants; and still others walk as the result of bone transplants. These are dramatic achievements, but they in no way reveal the complicating factors that must be investigated and eliminated prior to each progressive step.

When a technique was developed in this laboratory whereby fully developed second molars could be extracted and replaced in the hamster, a comparable intensive study involving the tooth became practicable. Since earlier work demonstrated that all necessary tissue elements (epithelial attachment, periodontal membrane, and pulp) were capable of regrowth,[1] and transplantation as well as replantation was feasible,[2] an analysis of some of the factors hindering or aiding this regrowth potential was prudent. If tooth tissue, that is, dentine, cementum, and enamel, could remain unaltered in chemical composition for an extended period of time, an artificial means of preservation would be unnecessary. If this were not the case, then a means of "banking" these tissues should be devised. The results to be reported in this article give some indication of how long teeth can be separated from their natural environment without detectable deviation in the results of regrowth.

REVIEW OF THE LITERATURE

It would be difficult to say who first attempted the transplantation of teeth. Lufkin[3] states, in his *History of Dentistry,* that transplantation of

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teeth, from the poor to the rich, was practiced in France as early as the seventeenth century. These reports, though, are vague and are mentioned here only to demonstrate that, even at that early stage of scientific development, the idea had been born. According to Wilkinson,[4] John Hunter transplanted a tooth into a cock's comb in 1855 and later, when the tooth was decalcified and opened, he observed blood vessels. Curtis[5] reported that he successfully transplanted a freshly extracted human tooth in 1888 and that a histologic section made some years later when the tooth was removed revealed viable odontoblasts.

When Fleming[6] transplanted human tooth germs to lower animals, "usually two to fourteen hours elapsed before these embryonic tissues were used," and they were refrigerated during this time. He stated that many of these tissues were accepted by their hosts and proceeded to mineralize and mature when transplanted. The outstanding features of the failures were either acute inflammation or gradual resorption. Shapiro[7] and Shapiro and Maclean[8] gave no time lapse between removal and transplantation of tooth germs in the cat.

Lefkowitz's[9] current work in growing tooth germs in tissue culture apparently utilizes freshly removed tooth germs.

There is also some literature concerning fully formed teeth. Wilkinson[4] extracted two lateral monkey incisors and replanted them five days later. Examination fifty-three days postoperatively showed no resorption of the alveolar bone but some root resorption. Hammer[10] replanted permanent teeth of young dogs after boiling the teeth in water for ten minutes. He removed the pulp, however, and filled the cavity with cement. The experiments ran from twenty to 179 days and showed considerable ankylosis of the roots. In 1955 Agnew and Fong[11] reported on a series of experiments then in progress using an unstated number of rhesus monkeys. They claimed transplantation of developing third molar teeth into the sockets of first molars. Since delay in transfer is not mentioned, it is assumed the operation was completed immediately following extraction. Microscopic findings after four days showed that "typical pulpal degenerative changes, especially fibroid, are taking place in areas most distant from apical contact with the socket granulation tissue." They reported that after four weeks the pulp appeared healthy, the periodontal membrane was approaching functional status, some osteodentine was seen lining the dentine walls, and differentiated odontoblasts were present. The two-year report has not been published.

Among the more recent attempts at clinical tooth transplantation, usually involving transplantation of an immature third molar into a first molar site, could be mentioned the following: Clark and associates,[12] basing most of their description on the histologic study after the transplantation, stated that some regrowth was obtained. Crain[13] reported the experimental reimplantation of an upper third molar. He washed the tooth and placed it in a 1:1,000 solution of benzalkonium chloride and stated that one hour and forty-five minutes elapsed before the tooth was implanted. After three weeks

the tooth was firm, comfortable, and in normal function. Collings[15] successfully transplanted two unerupted third molars to first molar sites. These teeth were wrapped in sterile gauze moistened with physiologic saline until ready for use. After twenty-five months the teeth were said to be functional. The length of time the teeth remained out of the socket in the above reports (except for Crain's[13] report) usually was not explicitly stated. Apparently the procedure of transplanting the third molar into the first molar site was done as a one-step operation; therefore, the time lapse probably would be in the nature of one hour or less.

It is evident that the only way to secure evidence concerning possible alterations in the tooth structure as a result of delay in its transplantation would be a comparison of regrowth results involving the same series of experiments, after different time intervals. It can be seen from the foregoing that none of the experiments on tooth transplantation to date has been organized in this fashion. The following report is the first attempt in this connection.

MATERIALS AND METHODS

For this investigation, the mandibular second molars were extracted bilaterally and replaced in their respective sockets in the following sequence of delay: thirty minutes, six hours, twenty-four hours, and forty-eight hours. A total of 146 teeth from seventy-three animals was used.

The surgical removal of the teeth was performed under light Nembutal sodium anesthesia (50 mg./c.c. solution), injected intraperitoneally, in the amount of 0.07 c.c. per animal. Each animal was 30 days old. Dental explorers fashioned into small elevators were used to expand the sockets, and curved Kelly hemostats served as extraction forceps for actual removal of the teeth.

For immediate replantation, the teeth were lifted from their sockets and blood was allowed to fill the space. The teeth were then returned and pressed firmly into place, and the animal was allowed to waken. Teeth that were to remain outside the mouth for the specified times before mentioned were placed in sterile physiologic saline at room temperature before they were returned to the sockets. These animals were, of necessity, re-anesthetized.

Following surgery, the animals resumed normal activity and a normal diet consisting of Purina Laboratory Chow, green vegetables, and water. After thirty days they were sacrificed by intracardiac injection of Nembutal sodium. The mandibles were removed and fixed in 10 per cent formalin for a period of two weeks. After electrolytic decalcification, alcohol dehydration, and paraffin embedding, 10 micra sagittal sections were cut, and slides made from every tenth section were stained with hematoxylin and eosin.

Quality of takes was graded 0 through plus 4, based on degree of pulp regeneration, regrowth of periodontal fibers, ability of the surface epithelium to attach at the cervical line, and severity of root resorption. Plus 1 and plus 2 were considered poor takes; plus 3 and plus 4 were considered good takes.

RESULTS

Percentage of successful takes in the immediate reimplants and thirty-minute delay series was essentially the same, with just over 50 per cent good takes in each group (Table I). The periodontal fibers were as well attached in one group as in the other, and root resorption was minimal to moderate throughout. In both groups, in most instances, the surface epithelium was attached at the tooth cervical line (Figs. 1 and 2). The attempt at pulpal regeneration was similar also. One possible difference in the two groups was noted—whereas very little inflammation was observed in and around the teeth that were replanted immediately (Fig. 3), occasional small abscesses were encountered in the apical region of the thirty-minute delays (Fig. 4). However, it was not possible to determine whether this occurred constantly enough to be significant in so limited a number of experiments. These abscesses, in most instances, were localized and seemed to be in the process of healing.

The histologic picture was changed completely in the six-hour delay group, and the twenty-four- and forty-eight-hour groups showed equally poor results (Table I). The dominant features in most of these animals were severe osteomyelitis, liquefaction of the soft tissues, and necrotic bone (Fig. 5). Forty-four per cent of the teeth were missing in the six-hour delay group and hence could not be evaluated. Of the teeth that remained, one or two showed fairly good attachment of the periodontal fibers (Fig. 6), but the surface epithelium in most instances seemed unable to adhere to the cervical portion of the tooth and migrated extensively (Fig. 7). The pulp tissue, for the most part, was necrotic, with no attempt at regeneration. Of the teeth that remained, only fractional root resorption was observed. Although all of the features of the six-hour group were not entirely negative, there were no good takes; therefore, the group as a whole was, of necessity, considered a failure.

TABLE I. COMPARISON OF RESULTS

TIME	NO. TEETH	NO. TEETH MISSING	TEETH MISSING (PER CENT)	POOR TAKES (PER CENT)	GOOD TAKES (PER CENT)
Immediate replants	34	0	0	41+	58+
30-minute delay	20	1	5	37	63
6-hour delay	30	15	44	100	0
24-hour delay	32	15	43	91	9
48-hour delay	30	8	26	94	6

The percentage of teeth missing in the twenty-four-hour group was 43 per cent, and of the remaining teeth 9 per cent were good takes. Again, severe tissue inflammation was encountered in many instances, as well as poor attachment of the periodontal fibers, extensive migration of the surface epithelium, and necrotic pulp, but the degree of root resorption was mild. In those experiments that did not show severe inflammation (9 per cent good takes), the pulp was necrotic, but there was good attachment (Fig. 8), little migration, and evidence of beginning repair around the apices; hence, these were considered to be good takes. In two of these groups an interesting

Fig. 1.—Perfect take of immediate replant. Low magnification showing viable pulp with odontoblasts, excellent attachment of periodontal fibers, no epithelial migration, and minimal root resorption.

Fig. 2.—Perfect take of tooth replanted after thirty minutes. Low magnification showing same features as Fig. 1.

Fig. 3.—Low magnification showing small amount of inflammation in crown of one side of immediately replanted tooth.

Fig. 4.—Low magnification of thirty-minute delay. Small abscess at apex, beginning granulation one side.

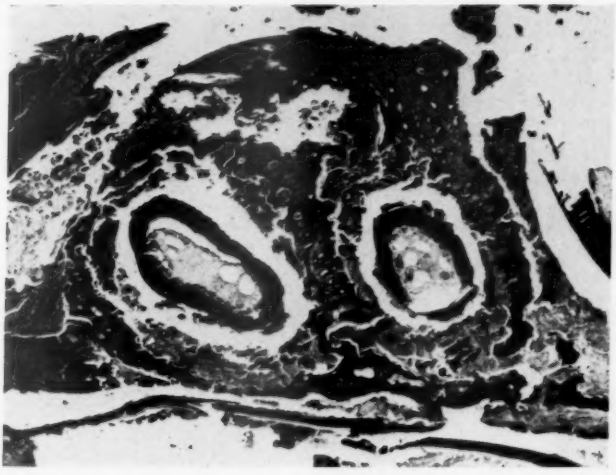

Fig. 5.—Low magnification of tooth replanted after forty-eight hours. Note osteomyelitis and liquefaction of soft tissues.

Fig. 6.—Low magnification of six-hour delay showing good attachment of periodontal membrane.

Fig. 7.—Low magnification of six-hour delay showing extensive migration of surface epithelium.

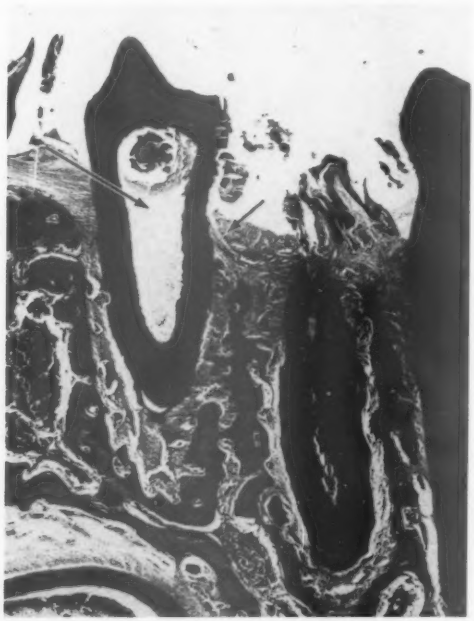

Fig. 8.—Low magnification of twenty-four-hour replant. Pulp is necrotic, but periodontal membrane is well attached, and epithelium has not migrated from the surface.

phenomenon occurred. An osteogenic mass the shape of the tooth was present in the area of the replanted tooth (Fig. 9). Inflammation was minimal in amount, and the tissues were otherwise quite healthy. Of course, no sweeping conclusions could be made from this finding, but the presence of such material only in the extended delay group might prove significant if pursued further.

Of the thirty teeth replanted after forty-eight hours, 26 per cent were missing and 6 per cent of the remaining teeth were good takes. Osteomyelitis, tissue inflammation, epithelial migration, and necrotic pulp were the main pathologic changes.

Fig. 9.—Tooth replanted after twenty-four hours showing osteoid material replacing tooth structure. Low magnification.

DISCUSSION

The fact that all of the teeth replanted immediately or after thirty minutes remained in the socket for the entire experimental period, with a minimum of tissue inflammation, as opposed to the loss of many teeth and a sharp increase of inflammation observed in the delayed series, would seem to indicate that some alteration either had developed or had been introduced. Since all of the teeth were kept at room temperature, it is possible that the waiting period of six hours or longer was sufficient for the incubation of large numbers of bacteria which, when introduced into the newly formed clot, produced an overwhelming infection that could not be overcome by the tissues. The surface epithelium and periodontal fibers may have been influenced by this factor also, since they seemed unable to reattach to the replanted tooth.

It is also possible that in the longer delay groups chemical alterations of the tooth substance could have occurred. Since the cementum and dentine contain appreciable amounts of organic material, their disintegration could

have produced chemicals that were no longer compatible with the host tissues. Of course, this study does not indicate whether this possible influence actually was operating.

It is recognized that some facets of the repair process could have been involved. Immediately after the extraction, obviously, the socket was filled with blood which clotted. The reintroduction of the tooth after six hours or more could have dislodged this clot without another forming. This, then, would have produced the equivalent of a dry socket. If the clot were not disturbed too severely, the early beginnings of the granulation process could have started, especially in the forty-eight-hour group. Whether this would be beneficial or detrimental was not demonstrated.

From the foregoing factors, it is evident that the entire story of the delayed replantation and the transplantation is not shown in this study. Each of the above factors individually needs to be studied intensely. However, this present study does seem to indicate that detrimental influences do become operative in a relatively short time.

CONCLUSIONS

1. Teeth can remain out of the oral cavity for as long as thirty minutes without deleterious effects, whereas delays of six hours or longer do show adverse results.

2. The above experiments would seem to indicate that replantation and transplantation procedures should be done as expeditiously as possible.

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PROFESSIONAL NEWS ITEMS

Postgraduate Courses at Boston University

The Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University, announces the following courses:

Periodontal Therapy, Oct. 6-17, 1958
Periodontal Pathology, Oct. 27–Nov. 1, 1958
Endodontics, Nov. 10-14, 1958
Occlusion and Occlusal Adjustment, Nov. 10-14, 1958
Crown and Bridge, Nov. 17-21, 1958
Partial Denture Principles, Design, and Construction, Jan. 8-10, 1959
Full Denture, Jan. 12-16, 1959
Oral Pathology (for oral surgeons), Jan. 12-16, 1959

Those interested may write to the Department of Stomatology, Division of Graduate Studies, School of Medicine, Office of the Dean, Boston University, 80 East Concord St., Boston 18, Massachusetts.

Crown and Bridge Course.—A course in fixed bridge prosthesis will be given Nov. 17 to 21, 1958, under the direction of Dr. Leo Talkov, Dr. David J. Baraban, and staff. By means of lectures, demonstrations, and student participation on models, emphasis will be placed on diagnosis, treatment planning, tooth preparation with high speeds, impression taking, fabrication of temporary acrylic restorations, and occlusal registrations—all coordinated with pertinent laboratory procedures to the finished bridge.

The fee for the course will be $175.00; enrollment is limited.

Removable Partial Denture Course.—A three-day postgraduate course in partial denture prosthesis will be given on Jan. 8, 9, and 10, 1959. This course will consist of lectures and demonstrations. Emphasis will be placed upon diagnosis, treatment planning, biomechanical principles of partial denture design, mouth preparation, precision attachments, laboratory procedures, and rebasing techniques. Students are invited to bring models and x-rays of problem cases for seminar discussion. The course will be conducted by Drs. David J. Baraban, Leo Talkov, and Chester Landy. The tuition fee is $175.00; enrollment is limited.

Full Denture Course.—A five-day postgraduate course in full denture prosthesis will be given Jan. 12 to 16, 1959. This course will consist of lectures and demonstrations. Emphasis will be placed upon the physiology, pathology, and surgical preparation of the edentulous ridge in addition to diagnosis, treatment planning, occlusion, and a technique for the construction of full dentures. This course is directed by Dr. Chester Landy. Enrollment is limited. The tuition fee is $150.00.

Oral Pathology Course for Oral Surgeons.—A one-week course in oral pathology will be given Jan. 12-16, 1959. The purpose of this course is to acquaint the oral surgeon with the tissue changes occurring in the various oral manifestations commonly encountered in the practice of oral surgery. The course will include lectures, seminars, and the projection of microscopic slides of the various conditions. The clinical aspect of the case will be discussed and correlated to the microscopic findings. It will be under the direction of Drs. Henry M. Goldman, Kurt H. Thoma, and Jack Bloom. The enrollment fee is $200.00.

American Academy of Oral Pathology

The annual meeting of the American Academy of Oral Pathology will be held April 17 to 19, 1959, at the University of Minnesota in Minneapolis, Minnesota. The Fellows and Council meeting is scheduled for April 17, the annual scientific meeting for April 18, and the seminar for April 19.

The examination for elevation to Fellowship in the Academy will be held on April 17, 1959, at the University of Minnesota. Applications for the fellowship examination may be obtained from the office of the secretary-treasurer. No applications will be accepted after Jan. 1, 1959.

Robert J. Gorlin, Secretary-Treasurer
University of Minnesota
Minneapolis 14, Minnesota

Thirty-Fourth Greater New York Dental Meeting

The Greater New York Dental Meeting, recognized as one of the largest in the world, will be held at the Hotel Statler, New York, New York, Dec. 8 to 12, 1958. This meeting is sponsored by the First and Second District Dental Societies under the direction of Dr. Adolph G. Wagner, General Chairman. Participants from all over the United States, Canada, and across the seas will bring the latest in professional techniques, scientific researches, and knowledge of diagnosis and treatment.

Some highlights of the meeting will be discussions of high-speed and ultrasonic techniques; dental health research exhibits from hospitals, colleges, and the Armed Services; and a television program through the courtesy of the Bureau of Medicine and Surgery of the U. S. Navy.

Further information may be obtained from Mrs. Mabel Purdy, Executive Secretary, Room 106A, Hotel Statler, New York 1, New York.

Armed Forces Institute of Pathology

The Oral Pathology Branch of the Armed Forces Institute of Pathology will present its annual course entitled "Pathology of the Oral Regions" March 23 to 27, 1959. This course is open to both government and civilian dentists. Dentists in the federal establishment should apply through appropriate channels and civilian dentists should apply to the address given below. Information as to content of the course will be given in a later announcement. There is no tuition, and applications should be made as soon as possible because of heavy registration.

The annual slide seminar, sponsored by the Registry of Oral Pathology of the American Dental Association and the Armed Forces Institute of Pathology, will be conducted by the Oral Pathology Branch of the Armed Forces Institute of Pathology on May 22, 1959. Approximately sixty can be accommodated. A slide set of twenty-five slides, together with a descriptive brochure, will be given each one attending for his permanent possession. This is a seminar on advanced oral pathology, and the specific subject will be "Tumors of the Maxillary and Mandibular Bones." Teachers and workers in oral pathology, oral diagnosis, and oral medicine will be given preference in the registration. There is no charge for this seminar.

Those desiring to register or wishing information regarding these course should write Colonel Joseph L. Bernier, Chief, Oral Pathology Branch, Armed Forces Institute of Pathology, Washington 25, D. C.

Tufts University School of Dental Medicine

The following postgraduate refresher courses are announced:

DPG. 602. Occlusal Equilibration. Feb. 9, 10, and 11, 1959. Tuition, $100.00. Dr. Edward T. Fischer, Assistant Professor of Periodontology.

DPG. 603. Occlusal Adjustment; Its Relation to Dental, Periodontal, and Temporomandibular Joint Problems. March 30 to April 1, 1959. Tuition, $100.00. Dr. Sigurd Ramfjord, Associate Professor of Periodontics, University of Michigan.

DPG. 605. A Special Participation Course in Periodontology. April 13 through April 17, 1959. Tuition, $150.00. Dr. Irving Glickman and staff.

DPG. 302. Local Anesthesia. Feb. 9 to 11, 1959. Tuition, $75.00. Dr. Leonard M. Monheim, Professor of Anesthesiology, University of Pittsburgh.

DPG. 301. Minor Oral Surgery. March 4 to April 22, 1959 (each Wednesday). Tuition, $150.00. Drs. Richard Taylor, Professor of Oral Surgery and Chairman of the Department, and Max Jacobs, Associate Professor.

DPG. 703. Full Denture Prosthesis. June 1 to 5, 1959. Tuition, $150.00; class limited. Drs. Irving R. Hardy, Professor of Prosthetic Dentistry (emeritus 1958), and N. Jack Hansen, United States Air Force.

For further information and application, write Division of Graduate and Postgraduate Studies, Tufts University School of Dental Medicine, 136 Harrison Ave., Boston, Massachusetts.

Earl Henry Memorial Clinic

The Second District Dental Society of Knoxville, Tennessee, and the University of Tennessee College of Dentistry wish to announce the twelfth annual Earl Henry Memorial Clinic to be held Oct. 17 and 18, 1958, at the University Center in Knoxville, Tennessee. This memorial clinic honors Dr. Earl Henry, who was the only Tennessee dentist killed in action in World War II.

The following subjects will be presented:

Practical Prosthodontics. Henry C. Parker, Charlotte, North Carolina.
Oral Surgical Procedures in General Practice. Thomas W. Jones, Birmingham, Alabama.

Those desiring to attend the Tennessee-Alabama football game on Saturday afternoon, October 18, should make arrangements for tickets with the respective University Athletic Association offices.

Sunland Seminar

The Greater Miami Chapter of Alpha Omega announces the premiere session of the Sunland Seminar, to be held Jan. 25 to 29, 1959, at the Deauville Hotel in Miami Beach, Florida.

The seminar, the theme of which will be "A Total Periodontal Concept," will be conducted by several of the foremost authorities in the country: Drs. Morton Amsterdam and Walter Cohen of Philadelphia, Donald McQueen of California, John Pritchard of Texas, and Balint Orban of Colorado.

Fee for the seminar is $250.00, which includes room (double occupancy) and modified American plan at the Deauville Hotel.

For further information and reservations, write to Dr. Irving Gordan, 420 Lincoln Rd., Miami Beach, Florida.

University of Alabama

During the month of December, 1958, the School of Dentistry will present the following refresher courses:

Oral Surgery for the General Practitioner.
Robert B. Shira, Colonel, DC, USA. December 6-8.

Preventive Orthodontics.
Sidney B. Finn, D.M.D., M.S. December 13-15.

For further information, please write to Director, Refresher Course Program, University of Alabama School of Dentistry, 1919 Seventh Ave. South, Birmingham, Alabama.

University of California Offers Research and Teaching Traineeships

In 1957 the School of Dentistry, University of California, San Francisco, instituted a research-teacher training program under the direction of Dr. Hermann Becks, Professor of Dental Medicine, with the financial support of the United States Public Health Service in Washington.

The program provides facilities and instruction in basic research and the principles of biology as they pertain to dentistry, including training in the fundamentals of animal experimentation, various types of operations, autopsies, intensive schooling in methods of tissue preparation, and a complete study of the gross and histologic results. It proides a good background for a teaching career and advanced scientific investigation as well as clinic practice. If the trainee is qualified and wishes to do so, he may take work leading to the M.S. and/or Ph.D. degrees. The annual stipend ranges from $4,000.00 to $6,000.00. This program is not limited to dental students or dentists but is open to graduate and undergraduate students in biochemistry, physiology, pathology, medical microbiology, and anatomy.

Further information may be obtained by writing to Dr. Hermann Becks, School of Dentistry, University of California, San Francisco 22, California.

University of Washington

A new course of graduate study in periodontics is announced. The course is of eighteen months' duration and, upon the satisfactory completion of a thesis in addition to the acceptable performance of the remainder of the academic program, leads to the granting of a Master of Science degree in dentistry.

The course will be directed toward the training of teachers and research workers in periodontics and allied fields. Extensive clinical training will be offered, in addition to the projection of a basic science background.

Inquiries should be addressed to Saul Schluger, D.D.S., Director, Graduate Dental Education, University of Washington, School of Dentistry, Seattle 5, Washington.

New York University

The New York University College of Dentistry is now offering fellowships and traineeships for graduate study. They are made available, in part, through research and training grants from the National Institute of Dental Research of the United States Public Health Service.

The graduate program for the fellows and trainees emphasizes basic science and research training. For the holder of the D.D.S. or D.M.D. degree, it also provides clinical correlation and the opportunity for advanced clinical training.

Applications will be received at any time and may be initiated by a letter to Dr. E. D. Goldsmith, research coordinator and training director, College of Dentistry, New York University, 421 First Ave., New York 10, New York.

University of Pennsylvania

The School of Dentistry announces the following postgraduate courses:

Dentistry for Handicapped Children. Dec. 1 to 3, 1958.
Complete Denture Prosthesis. Jan. 12 to 15, 1959. Dr. Vincent R. Trapozzano.
Hospital Dentistry. Jan. 26 to 28, 1959. Dr. John P. Looby and associates.
Prosthetic Relations of the Temporomandibular Joint. Jan. 19 to 23, 1959. Dr. Ernest R. Granger.

For further information, please write to Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pennsylvania.

University of Minnesota

The second annual course in advanced oral pathology will be given during the week of April 20 to 24, 1959. The course consists of both lectures and laboratory work. The subjects and faculty will be as follows:

Tumors of the Skin. Dr. Herbert Z. Lund.
Tumors of Soft Tissues. Dr. Raffaele Lattes.
Skin Dermatoses. Dr. Robert Goltz.
Non-neoplastic Diseases of the Jaws. Dr. William Shafer.
Tumors of Bone. Dr. David C. Dahlin.
Radiation Pathology. Dr. Donn Mosser.
Advanced Roentgenographic Diagnosis. Dr. Edward Stafne.
Salivary Gland Pathology. Dr. Anand Chaudhry.
Odontogenic Lesions. Dr. Robert Gorlin.

The tuition will be $65.00. The course is limited to fifteen persons. Housing for the five days will be approximately $15.00. Those interested should immediately contact Dr. Robert J. Gorlin, Chairman, Division of Oral Pathology, School of Dentistry, 242 Owre Hall, University of Minnesota, Minneapolis 14, Minnesota.

REVIEWS OF NEW BOOKS

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Motivating Patients for More Effective Dental Service. By J. Lewis Bloss, PhG., D.D.S., Professor of Periodontia and Oral Medicine, Lecturer in Practice Management, New York University College of Dentistry. With contributions by Ben B. Drob, D.D.S., Reuben Fogelson, D.D.S., Alice Gustav, A.B., A.M., Ph.D., and William Wolf, B.S., M.D., Ph.D., Philadelphia, J. B. Lippincott Company. Price, $6.50.

As stated in the preface, "The book is not text-book writing, it is instead a congenial discussion of the subjects pertinent to all dentists who endeavor to achieve an effective and personally rewarding practice."

This reviewer enjoyed reading the book and feels that the young dentist, the secretary, and office assistant would benefit from reading it.

T. J. C.

Cleft Palate and Speech. By Muriel E. Morley, Speech Therapist-in-charge of the Speech Therapy Unit, The United Newcastle upon Tyne Teaching Hospitals and the Newcastle upon Tyne Hospital Management Committee Group. Baltimore, Williams & Wilkins Company. 271 pages, 86 illustrations. Price, $6.50.

When the first edition of this text was published, Miss Morley made fine contributions toward an understanding between the surgeon and the speech therapist.

In the fourth edition the roles of the orthodontist and the prosthodontist, along with further advances in surgical technique, are stressed.

The book is recommended to those who are interested in the management of cleft palate and associated deformities.

T. J. C.